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CERVICAL DISKOGRAPHY

TECHNIQUE, INDICATIONS AND USE IN DIAGNOSIS OF RUPTURED CERVICAL DISKS

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THE roentgenographic visualization of the intervertebral disk by injection of a radiopaque solution directly into the disk was first introduced by Schmorl and Junghanns.[17] In their classic monograph on the studies of the intervertebral disk published in 1932, dyes and opaque substances such as red lead were injected into the nucleus pulposus for roentgenographic and photographic visualization. Lindblom[12,13] (1941, 1944) made anterior injections of red lead into the disk to demonstrate radial ruptures of the annulus fibrosus. In 1948, Lindblom[14] first described the clinical use of diskography for roentgenographic demonstration of the normal and pathologic anatomy of the intervertebral disk. Since then diskography of the lumbar intervertebral disk has had wide usage, and numerous publications, particularly in the foreign literature, have evidenced its clinical value. The internal rupture of a lumbar intervertebral disk following injury may be the cause of the symptom of low back pain without sciatica. This lesion is graphically demonstrated by the diskogram in the presence of a normal myelogram.[3]

The value of lumbar diskography as a diagnostic method was recognized early[3] and adopted as a routine procedure in the clinical study of the pathology of the intervertebral disk.[4] We have followed closely a group of over 400 patients upon whom lumbar diskography had been performed in the past six years. The conclusions drawn from this volume of experience are: if properly performed a normal disk cannot be injured by needle puncture and injection; more diagnostic information can be obtained by diskography than myelography; pain caused by disk injection is a valuable objective sign in localizing the abnormal disk from which the symptoms originate. The application of these advantages of lumbar diskography to the investigation of ruptured cervical disks has been greatly needed and is long overdue.

Injuries to the intervertebral disk occur primarily in the lumbar and cervical spine in the ratio of approximately 30:1.[7] The clinical syndromes resulting from rupture of a cervical disk with herniation into the spinal canal is well known. The early contributions of Stookey[19] and Stookey,[20] Bucy *et al.*,[2] Semmes and Murphey,[18] and more recently the extensive studies made by

Swedish neurosurgeons[8] have contributed to our knowledge of the pathologic physiology of ruptured cervical disk. These writers have described the cervical nerve root syndrome causing symptoms of neck pain with radiation into the precordium, the region of the scapula, the shoulder and the upper extremity with paresthesias of the fingers and the cord syndrome with spasticity in the lower extremities. Recent renewed interest in the subject of ruptured cervical disk has been stimulated by injuries to the cervical spine —the so-called "whiplash injury."[10] This term coined by the legal profession has become a popular motive for damage suit and litigation following automobile accidents. The clinical investigation of these patients who complain bitterly of pain in the neck following snapping injury to the cervical spine has stimulated the investigation of the cervical intervertebral disk as a possible source of these symptoms.

The possibility of studying by disk injection, the referred pains in the neck, shoulder and arm originating from the cervical intervertebral disks has been considered for some time. Performing a diskogram on the cervical intervertebral disks was not possible if the method employed for lumbar diskography was used. The posterior approach to the lumbar intervertebral disk through the spinal canal and the roots of the cauda equina is a safe and comparatively easy procedure. Such an approach to the cervical intervertebral disk would, of course, be impossible since the needle would pass through the spinal cord. The first cervical diskogram, to my knowledge, was performed in 1955 by Walker[21] of Atlanta, Georgia, and shortly thereafter came into regular use in our clinic. The technique of disk puncture by the anterolateral approach was first done on 10 fresh cadavers and the roentgenographic configuration of the cervical disk visualized after injection of a radiopaque solution was studied. A series of 41 clinical cases with symptoms referable to the cervical spine have been studied by this method in the past eighteen months with interesting results.[5] Although our experience is limited, the information accumulated to the present time would indicate that cervical diskography has considerable diagnostic value, both clinical and roentgenologic and justifies a preliminary report.

Before giving the details of the technique of cervical diskography, it is necessary to list and describe several items of equipment required for this method. Since the procedure is carried out with the patient in the supine position, a special roentgenographic box must be made (Fig. 1—B). The box is placed beneath the head and shoulders and holds the roentgen-ray cassette in a vertical position for the lateral roentgenogram and horizontally in a tunnel beneath the neck for the anteroposterior projection (Fig. 1). The box measures 19 inches wide, 17 inches long and 2½ inches high; is wedge-shaped with the apex of the wedge beneath the shoulders. The top surface of the box has an opening 5½ inches wide and 11 inches long which is covered with a removable piece of plastic (lucite) ½ inch thick. The remainder of the top surface is built of two layers of ⅜ inch plywood with ⅛ inch of lead between them. The tunnel into which the cassette is placed for the anteroposterior roentgenogram will accommodate a 10 by 12 inch cassette. The cassette may be placed in the center beneath the plastic opening for a single anteroposterior view or may be moved from one side of the tunnel and then to the opposite side to obtain two views on the same film for stereoscopic projection. The roentgen tube is moved in a transverse direction. On the left side of the box is a small vertical slot 1¾ inches deep and 13 inches long and just wide enough to permit an 8 by 10 inch grid cassette to be inserted in a vertical plane for the lateral projection. The disk puncture is normally made on the right side diagonally into the center of the disk (Fig. 1—N). If the operator is left handed, however, the puncture can be made from the left and the slot in the box for the vertical projection can be made on the opposite side. The box is

slipped beneath the patient's head and shoulders. If it is uncomfortable for the patient to lie upon, a wedge-shaped sponge may be placed over the lower border of the box for padding (Fig. 1—S). The head and neck are placed in a position directly over the center of the plastic window. The head may be secured to the box with an adhesive strap or just kept in position throughout the procedure by the patient. The latter was found to be preferable in cooperative patients as the procedure is done under local anesthesia. Two roentgen-ray machines are used (Fig. 1—X). If the procedure is carried out in the roentgen-ray room, the patient may be placed on a roentgenographic table using the overhead roentgen-ray unit for the anteroposterior projection and a portable machine for the lateral, or the two portable machines may be utilized. The preparation tray for cervical diskography consists of antiseptic solutions for preparing the skin of the neck; 1 per cent procaine solution, a 2 cc. syringe and a tuberculin syringe preferably with a Luer lock attachment; 1 cc. ampule of 50 per cent hypaque or 70 per cent urokon, and the diskogram needles. Two sets of 2 needles are kept sterile in a separate large test tube. The shorter needle is either a No. 20 or No. 22 having a moderately long bevel and measuring 2 inches in length. The finer needle which passes through the larger one is a No. 25 or No. 26 caliber needle and is $2\frac{1}{2}$ inches long.

Fig. 1. Photograph of cervical diskography apparatus. B—wedge-shaped, lead lined box to hold two cassettes; S—radiolucent sponge beneath head and shoulders; N—diskography needles—anterolateral disk puncture; X—two roentgen-ray machines used, one for anteroposterior and one for lateral projection.

TECHNIQUE

The patient is placed in position on the cervical roentgenographic box and a preliminary test set of anteroposterior and lateral roentgenograms are obtained. A small lead marker for localization is placed on the neck directly over the disk to be punctured. In the patient with a short thick neck, it may be difficult to visualize the lower cervical disks because in the supine position the shoulders often overshadow the spine. This can be determined in the preliminary roentgenogram. If the fifth, sixth and seventh cervical interspaces are not well visualized, the procedure cannot be carried out unless the patient's shoulders are drawn downward. The patient can voluntarily lower his shoulders or they can be pulled down by an assistant.

In preparation for the diskography the patient is routinely given $\frac{1}{6}$ grain of morphine, should the procedure be painful. In apprehensive patients mild sedation with barbiturates may be advisable. The neck is prepared with antiseptic solution and novocaine is injected into the skin with a fine needle at the site of the disk puncture. The operator's finger tips are then placed in the groove between the carotid sheath and the trachea and gently pressed down to the vertebral body. The fine hypodermic needle is advanced through the tissues of

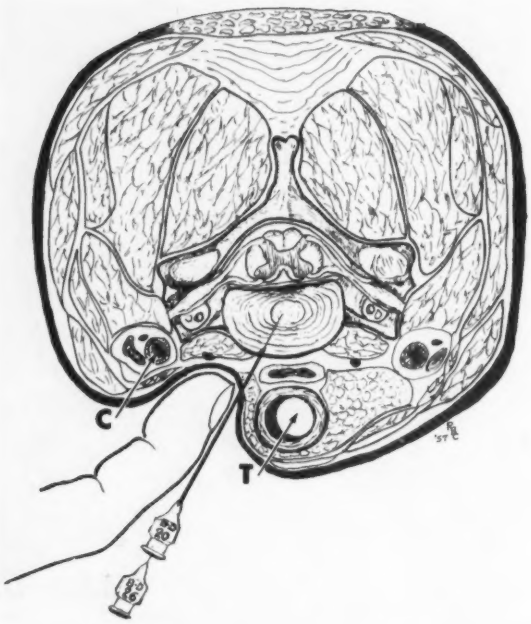

Fig. 2. Diagram to demonstrate anterolateral approach to the cervical intervertebral disk with digital pressure between carotid artery (C) and trachea (T).

the neck until it makes contact with the surface of the vertebral body and approximately 5 cc. of novocaine injected. This anesthetizes the periosteum of the anterior longitudinal ligaments, the prevertebral fascia and anterior annulus of the disk to relieve pain which may result from deep palpation with the finger tip in locating the disk or from the insertion of the diskographic needle.

When the tissues are well anesthetized, it is possible to feel the anterior surface of the intervertebral disk and the body of the cervical vertebra with the finger tips by displacing the trachea and esophagus medially and making firm pressure against the cervical spine. The humps of the intervertebral disks can often be palpated. This is easier in older patients in whom the margins of the vertebral bodies may be hypertrophied. Since most of the cervical disk pathology involves principally the two lower cervical disks, C5–6 and C6–7, these disks are usually studied. The fifth intervertebral disk is palpated between the inferior border of the thyroid and cricoid cartilages. The sixth disk is approximately 1½ cm. below it.

When the disk has been located by palpation, the operator's index and middle fingers are placed over it in the vertical plane and pressed tightly against the anterior surface of the vertebral bodies. This pushes aside the soft tissues of the neck so that when the first needle is passed through the skin, the vertebral body and disk are encountered at a depth of less than 1 cm. (Fig. 2). The first needle, a 2 inch No. 20 guide needle, is passed through the skin. If it is not possible to palpate the disk with the finger tips, the needle will encounter bone. The needle is then "walked" up and down, making individual light stabs into the bone of the vertebral body until the soft disk is encountered. Because of the looseness of the soft tissues of the neck, the needle may be moved over 1 cm. upward or downward in the vertical plane through a single skin puncture until the anterior surface of the disk is found. The needle is then inserted 1 or 2 mm. through the anterior longitudinal ligament and the anterior fibers of the annulus fibrosus. The palpating fingers are removed and the soft tissues of the neck are permitted to expand backward about the guide needle. At this point the patient is told not to talk, cough or swallow since the movements of the neck muscles through which the needle passes may dislodge it from the disk. A lateral roentgenogram is obtained to determine the position and direction of the first needle (Fig. 3*A*). If the roentgenogram shows that the needle point has entered the anterior surface of the correct disk and is in the proper plane, the second fine needle is passed through the larger needle into the nucleus pulposus (Fig. 3*B*). Since the fine needle is only 1 cm. longer than the larger one, there is no danger of advancing it too far (through the disk) providing the point of the first needle has entered the disk only 1 or 2 mm.

This first part of the procedure is carried out with the patient's occiput on the

plastic window of the cassette box. The neck is in slight hyperextension to open up the interspace anteriorly and permit easier puncture. When both needles are in place, the head is carefully elevated and a small radiolucent sponge is placed under the head to straighten out the cervical spine. The position of the needles is then checked, with a lateral and anteroposterior roentgenogram to determine their position before proceeding with the injection (Fig. 3, *A* and *B*, Fig. 6*A* and Fig. 7*B*). The anteroposterior film is essential to determine the position of the needles in relation to the center of the disk. Since the needles are inserted into the disk in a diagonal direction, it is possible for the point of the fine needle to be placed beyond the nucleus and into the fibers of the annulus lateral to the midline. If this occurs, the injection of the opaque material into a normal disk is impossible. The roentgen tube is angulated upward 10 degrees so that the rays are parallel to the plane of the vertebral bodies. If more than one disk is to be studied, a second needle may be inserted into an adjacent disk in a similar manner before the injections are made (Fig. 6*B*).

When the second group of roentgenograms demonstrates the point of the needle in the region of the nucleus pulposus, the opaque solution is injected. The amount of solution which could be injected into a cervical disk is surprising. It is customary to inject relatively large amounts of 35 per cent diodrast into a degenerated lumbar disk, often as much as 5 to 6 cc.; therefore, the cervical disks in the first cadavers were forcefully injected with quantities up to 1 cc. The roentgenograms demonstrated the opaque solution to be outside the intervertebral space, either in the spinal canal or the anterior paravertebral regions, apparently escaping through defects in the longitudinal ligaments. The normal nucleus pulposus of a cervical disk will accept only 0.2 cc. to 0.3 cc. of injected solution. The maximum which should be injected into a ruptured or degenerated disk is 0.5 cc. This finding corresponds to the measurements

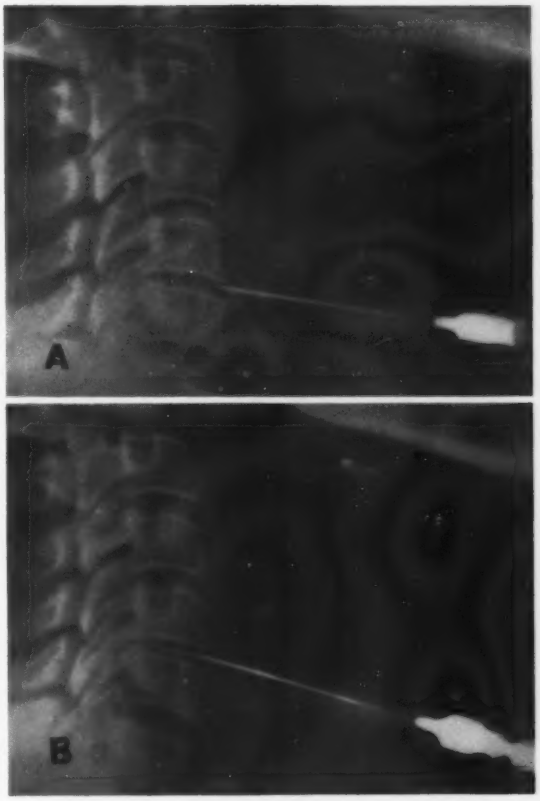

Fig. 3. Cervical diskography technique. (*A*) Lateral roentgenogram to show guide needle (No. 20, 2 inches long) with point inserted into the anterior fibers of annulus fibrosus. (*B*) Fine needle (No. 26, 2½ inches long) into the nucleus pulposus. Anteroposterior roentgenograms demonstrate the fine needle in the center of the transverse diameter of the disk (see Figures 6*A* and 7*B*).

made by Brain, Knight and Bull[1] who found that the nucleus pulposus comprises 15 per cent of the volume of a disk which in the cervical disks amounts to 0.21 cc.

The opaque solution used is either 50 per cent hypaque or 70 per cent urokon. Diodrast (35 per cent) in quantities of 0.1 to 0.2 cc. is not roentgenographically visible. A 1 cc. tuberculin syringe is used, preferably with a Luer lock tip to prevent the syringe from slipping off the needle. The injection is made slowly while holding the needles with the left hand to prevent dislodging them farther into the disk. During the injection the patient is asked to report any pain or discomfort and to localize the areas in which the pain is felt.

Fig. 4. Cervical diskogram. Whiplash (flexion) injury. Posterior rupture of annulus fibrosus C5–6 disk. Normal nucleus pulposus C6–7 disk below.

The syringe is removed and lateral and anteroposterior roentgenograms are taken with the needles in position. Stereoscopic exposures are useful in studying cervical diskograms because often small amounts of the opaque material can be localized more easily if seen in the third dimension. The patient's head is kept stationary in one position throughout the entire procedure. A "hot tank" for rapid film developing and fixing will speed up the procedure considerably. The films are examined, and if they are not satisfactory additional opaque solution is injected. If the films are satisfactory, the needles are removed and an additional set of roentgenograms taken without the needles. One may wish to use oblique views to better demonstrate unilateral protrusions into the intervertebral foramen. The roentgenograms must be taken as soon as possible after injection because the small amount of contrast medium may diffuse or absorb from the disk in a very short time. If a unilateral disk protrusion is suspected, as evidenced by symptoms and signs in one upper extremity, oblique roentgenograms are obtained. The roentgen tube may be angulated laterally 45 degrees, keeping the patient's head in the same anteroposterior position or the patient's head and trunk may be turned for oblique exposures, which usually are made on both sides for comparison.

DISKOGRAMS

The interpretation of the roentgenographic findings in the cervical diskograms has not been difficult. There are three types of pathologic cervical disks which may cause clinical symptoms. The first, and perhaps the one from which most valuable information can be obtained by diskography, is the early disk rupture, the so-called soft disk. This is the lesion which one would expect to find in the whiplash injury (Fig. 4). Plain roentgenograms of the cervical spine will show no demonstrable narrowing of the intervertebral spaces and no evidence of bone proliferation about the margins of the vertebral bodies. The only roentgenographic finding indicative of a cervical disk lesion may be the absence of the normal lordotic curve of the cervical spine, the so-called poker spine, or actual reversal of the normal cervical curve with the apex of the curve at the level of the lesion. In these cases, the diskogram demonstrates a posterior rupture of the fibers of the annulus fibrosus permitting extrusion of the opaque solution beyond the posterior limits of the nucleus pulposus (Fig. 5). In the lateral roentgenogram, the opaque material forms a small hump on the posterior surface of the intervertebral disk, indicating an extrusion of the nucleus pulposus backward into the spinal canal. In the anteroposterior roentgenogram the opaque solution may be seen

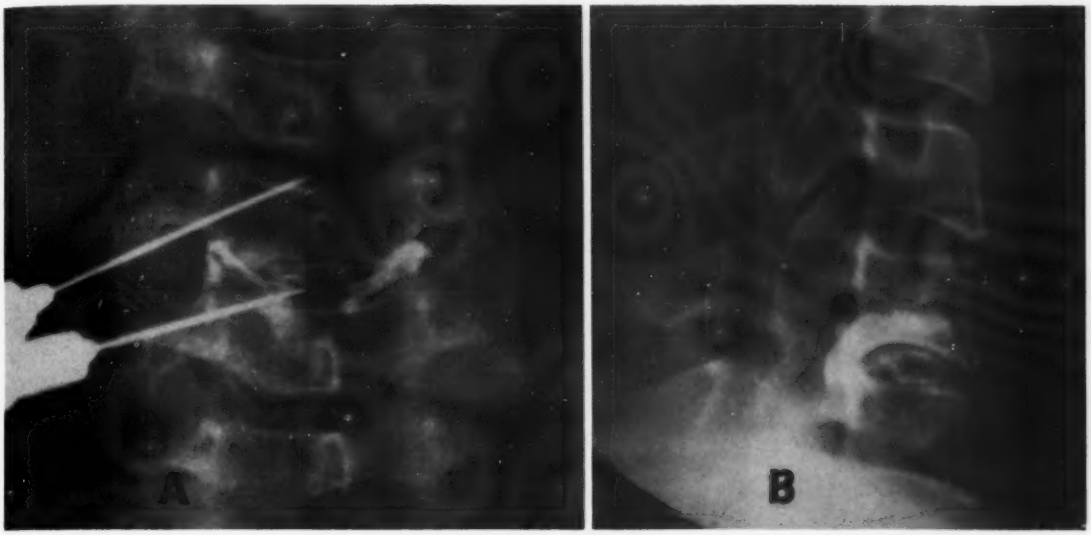

Fig. 5. (*A* and *B*) Diskogram of acute rupture C5–6 (three months after whiplash injury). Severe neck pain with bilateral shoulder and arm radiation, reproduced by disk injection. Roentgenogram of spine normal. Diskogram shows extensive bilateral and posterior disk laceration.

to extend into the lateral recesses of the disk, either unilaterally (Fig. 6*A*), or bilaterally (Fig. 6*B*), whereas in the normal disk it remains confined to the nucleus pulposus in the midline (Fig. 6*B*).

The second type of disk pathology demonstrated by the roentgenogram is the degenerated disk of long standing. These lesions are readily demonstrable in the plain roentgenograms by narrowing of the intervertebral space. Proliferation of bone about the margins of the vertebral bodies resulting in spur formation may be present. These osteophytes seen in the lateral projection may protrude posteriorly as well as anteriorly, encroaching upon the spinal canal in the midline. Oblique roentgenograms may demonstrate the encroachment of these osteophytes upon the intervertebral foramen. Clinically, the proliferation of bone posteriorly encroaching upon the spinal canal often results in serious impair-

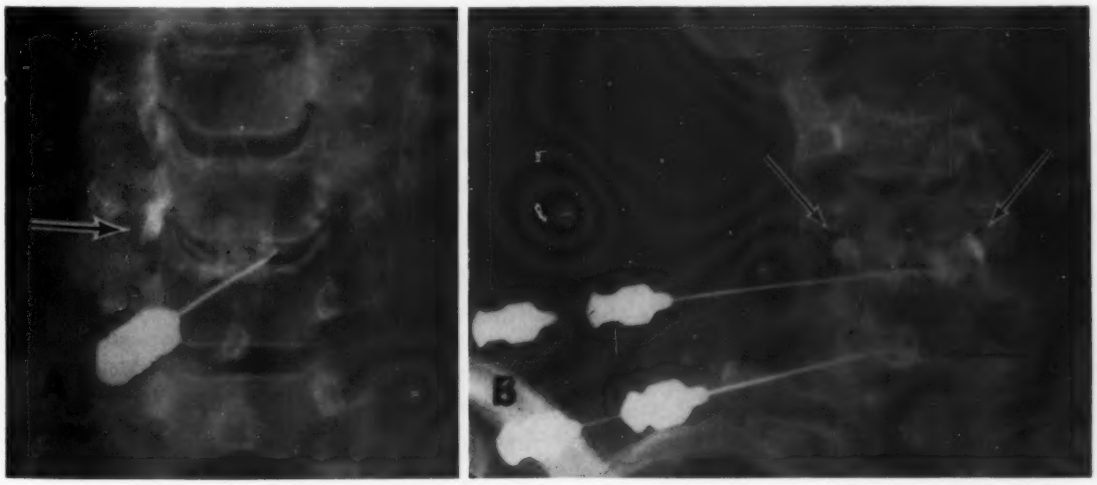

Fig. 6. Anteroposterior diskograms of acute lateral C5–6 disk herniations. (*A*) Unilateral, (*B*) bilateral (white arrows). Normal disk at C6–7 (*B*, black arrow). Double needle technique.

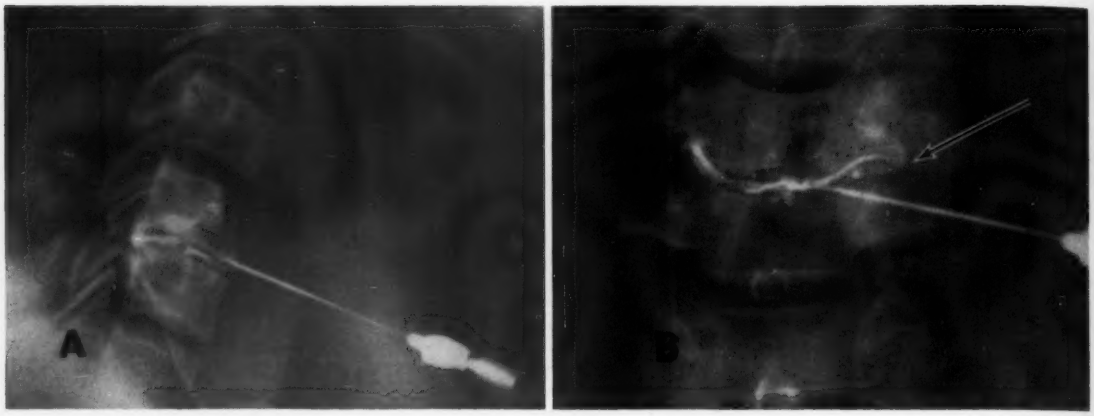

Fig. 7. Cervical diskogram of degenerated intervertebral disk without osteophytes. Note that opaque solution fills entire interspace (*A*) and extends well beyond limits visualized by myelography (*B*, arrow).

ment of function of the adjacent nerve roots, anterior horn cells or pyramidal tracts of the spinal cord. Severe brachial neuralgias, flaccid paralysis of deltoid and biceps muscles and more serious spastic paralysis of the lower extremities are frequently encountered in neurologic syndromes associated with these diskogenic bone overgrowths of the cervical spine. One may say, and justifiably so, that a diskogram in the presence of an obvious lesion demonstrated by the plain roentgenogram is superfluous. However diskographic findings in these patients have often demonstrated soft tissue lesions within the spinal canal much longer than would have been anticipated from the plain roentgenograms or the myelogram (Fig. 7).

The third type of abnormal cervical diskogram is the massive disk protrusion into the spinal canal. These lesions are often diagnosed as cervical cord neoplasms and show obstruction of the spinal canal by myelography. The possibility of making a differential diagnosis between a neoplasm and a herniation of the intervertebral disk by cervical diskography was demonstrated in a recent case (Fig. 9).[1] After the opaque solution was injected into the disk, the roentgenograms demonstrated a large hemispherical mass on the anterior wall of the spinal canal obviously communicating with the intervertebral space and verified at operation as a large herniated disk.

In most patients some pain was experienced by the disk injection. The character and location of the pain varies with the pathologic changes in the disk. In a normal disk, that is, one in which the roentgenogram shows a normal interspace, there may be no discomfort or if pain does occur from the injection it is localized posteriorly in the midline at the base of the neck. This same localization was described by 4 patients in whom normal disks were injected, 1 at C_4, 2 at C_5 and 1 at C6.

In the patients having pathologic disks suspected either by the clinical signs or roentgenographic findings, pain was produced by diskography. When a unilateral disk protrusion was suspected clinically by a unilateral brachial neuralgia intensified by movements of the neck, the disk injection caused severe pain at the base of the neck with unilateral radiation into the scapula, over the shoulder and into the upper arm. In patients having subjective numbness of the fingers, some paresthesia of the hand was observed with the intensified pain resulting from the injection. However, in most patients with a unilateral nerve root syndrome, the pain from disk injection did not extend below the elbow. In those patients whose clinical picture indicated spinal cord involvement, the pain experienced by disk injection also included a shock like sensation "like electricity" which spread down the middle of the spine

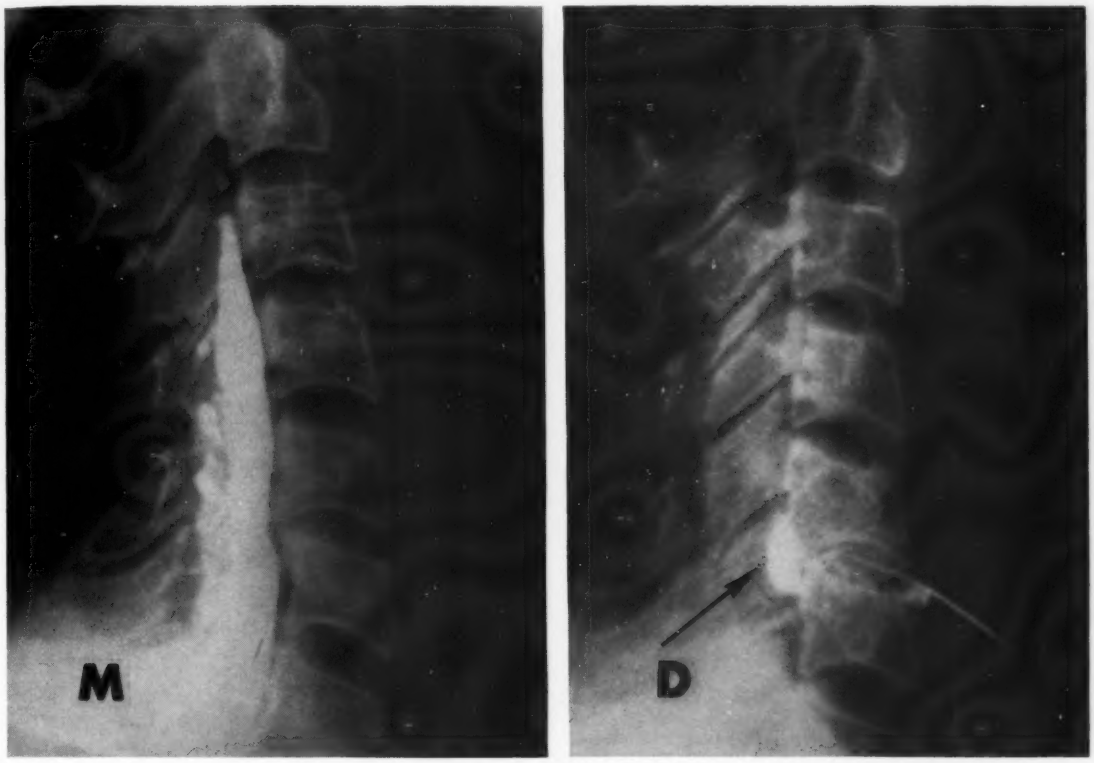

Fig. 8. Early degeneration of C5–6 intervertebral disk with mild posterior osteophyte formation. Myelogram (M) does not disclose the size of the large midline disk protrusion demonstrated by the diskogram (D, arrow).

as far as the coccyx in 1 patient, and all the way to the toes in another. Almost all patients described some pain at the base of the neck. None have had precordial pain although inquiry has been made in each case.

The pain most frequently experienced by cervical disk injection is referred to the region of the scapula, usually unilaterally, but may be bilateral and may originate either in the fifth or sixth cervical disk. This pain has been observed by most patients when the first needle puncture was made in the disk, indicating that the nerves in the anterior longitudinal ligament and anterior annulus, when irritated, will refer pain to the back in the region of the vertebral border of the scapula. The pain is often intense but eases off and disappears within two to five minutes. These brief experiences suggest that disk injection for the cervical diskogram may give valuable diagnostic information in determining the origin of referred pains from the disk to the neck, shoulder and arm, similar to referred pain from the lumbar disks. More experience with this procedure will be necessary, however, before positive localization of the exact origin of these pains can be determined; that is, which are neurogenic and which are diskogenic pains.[5]

Whenever the subject of diskography is discussed, severe objections are frequently raised because of the possible danger of damaging a normal intervertebral disk by puncturing it with a needle and injecting material into it. It is to be expected that such objections will be raised when the subject of cervical diskography is proposed. After six years of experience performing diskography on the lumbar intervertebral disks, we have yet to encounter a single patient in whom it was suspected that the puncture and injection of a normal intervertebral disk resulted in symptoms of disk rupture at a later date. This statement may have more significance if it is pointed out

Fig. 9. Roentgenogram of patient diagnosed clinically and by myelography as a spinal cord tumor. Shown by cervical diskography to have a massive intraspinal herniation of intervertebral disk.

that all of our patients are situated on a small island surrounded by thousands of miles of water and few of them leave to go elsewhere. If any trouble results from procedures on their spine, the doctor is immediately informed of it. Anatomically, it does not seem logical to assume that the passage of a fine needle through the tough interlacing fibers of an intact annulus fibrosus could do harm or damage to them. These structures are daily subjected to tremendous traumatic insults in the normal activities of the spine. A fine No. 25 needle passed through these tough fibers by a single puncture could not possibly tear or weaken them to the point of causing a disk to rupture at a later date as some have suggested.[11]

Secondly, the injection of a solution into the nucleus pulposus of a normal disk has also been criticized. It has been stated that such an injection may result in necrosis or destruction of the interior of the disk.[15] This criticism is also unjustifiable. The solution to visualize the lumbar nucleus pulposus is 35 per cent diodrast. This material is regularly used for roentgenographic demonstration of the kidneys and arteries. Also a small quantity is routinely placed in the conjunctival sac of the eye to determine sensitivity. Any "foreign" material which can safely be injected intravenously for pyelography, intra-arterially for cerebral angiography or placed in the eye without danger of irritation to its delicate tissues should be considered safe to inject into an intervertebral disk. Also the chemical make-up of the intervertebral disk with its high concentration of ground substance, particularly hyaluronic acid[9] and its active diffusion of fluids, is conducive to rapid removal of the fluids injected into it. It has been frequently demonstrated in a normal lumbar disk that the opaque material has diffused through the disk into the body of the vertebra and thus into the venous circulation as rapidly as fifteen minutes after injections. These facts should certainly dispel the fears and criticisms of those who are reluctant to accept diskography as a valuable diagnostic procedure on the basis of injury to a normal intervertebral disk.

The possibility of damaging the more delicate cervical disk by disk puncture and diskography may also be considered. Injury to the cervical disk by the technique herein described is probably less likely than that of the lumbar disk because of the location of the disk puncture. In the lumbar region, the disk is entered with the needle through the midline, posteriorly. This region is known to be the weakest part of the lumbar disk, the region in which the fibers of the annulus fibrosus are the thinnest and the site at which pathologic ruptures most frequently occur. The cervical diskogram is performed by inserting the needle into the anterolateral margin of the disk. The fibers of the annulus fibrosus in this region are nearly twice as thick and heavy as those posterior to the nucleus. The danger of

damaging these tough annular fibers in the cervical disk is therefore less likely. It has also been proposed that the forceful injection of the contrast medium into the nucleus pulposus could possibly rupture the fibers of the annulus fibrosus. This, of course, is physically impossible. In the lumbar region, the noncompressible nucleus pulposus of the intervertebral disk exerts static pressure on the surrounding annulus of well over 100 pounds in the upright position. It would be impossible to apply a force comparable to this with a syringe through a fine needle. Likewise, in the cervical region, although the compression force upon the small cervical disk is considerably less than that on the lumbar disk, the injection of the disk with a fine needle and a 1 cc. tuberculin syringe likewise does not compare with the weight of the head and neck which the lower cervical disks normally support.

One cannot discuss diskography without mentioning its value in comparison to myelography. Demonstration of a pathologic lumbar intervertebral disk by diskography from which the symptoms of low back pain originate is very frequently encountered in the presence of a normal myelogram. Likewise, posterolateral herniation of disk fragments causing severe nerve root compression is frequently associated with a normal myelogram when the herniation is lateral to the dural sac and the short dural sleeve of the nerve root and therefore beyond the column of intradural opaque oil. This is particularly true at the lumbosacral level and a comparable situation is found in the cervical spine.

The frequency with which the cervical myelogram has failed to demonstrate the presence of a herniated cervical disk in patients whose clinical picture indicates an obvious nerve root lesion has discouraged some surgeons from doing cervical myelography in suspected disk lesions. The clinical signs and symptoms and the neurologic findings have been more dependable criteria for surgical exploration of the nerve root than myelography. Our limited experience with cervical diskography would indicate that more diagnostic information can be obtained through this roentgenographic medium in cases of this type. In the anteroposterior roentgenograms, opaque material injected into the disk is shown to extend into the lateral recess of the disk beyond the limits of the dural sac visualized on the myelogram (Fig. 6*A*). Likewise midline disk herniations may not show on a myelogram or may appear as a small notch in the oil column in the lateral projection (Fig. 8—M). This is due to the oil filling the space on either side of the midline protrusion. Diskography in these patients gives a more accurate picture of the exact location and size of the protruding lesion (Fig. 8—D).

In the severe brachial neuralgias, localization of the nerve root involved can often be determined by the neurologic signs and symptoms. The disk protrusion between the fifth and sixth cervical vertebrae usually causes pain and paresthesias radiating into the thumb and index fingers. But the pain from the C6–7 disks may radiate either along the radial or ulnar nerve distribution. Frequently, the clinical picture is not sufficiently clear for exact localization. A recent patient with large osteophytes at C6–7 only had thumb and index finger paresthesias. The C_5 disk was normal. The pain may be of a diffuse character involving the entire arm and hand. A differential diagnosis must therefore be considered between neuralgias from other causes such as the scalenus anticus syndrome and other lower brachial plexus or peripheral nerve pathology.[16] It is in this realm of differential diagnosis that cervical diskography may prove most helpful. By injecting the cervical disk, the intra-disk pressure may be sufficiently increased to displace the herniated disk fragment against the involved nerve root and the patient's brachial pain intensified or reproduced. An accurate localization of the level of the pathology can thus be determined as well as the demonstration of the presence of a disk lesion rather than some other peripheral nerve,

brachial plexus or neck pathology to account for the symptoms.

SUMMARY AND CONCLUSION

A report on experience with the roentgenographic visualization of the cervical disks by diskography is presented. The technique of disk puncture by the anterolateral approach for injection of the nucleus pulposus is described. The roentgenographic interpretation of the normal and abnormal cervical disk is based upon experience with 41 clinical cases upon which cervical diskography had been performed over an eighteen month period.

It is concluded that cervical diskography is a relatively simple and safe procedure. It may offer diagnostic advantages over other methods used for detecting the presence of pathology in the cervical region to account for neurologic syndromes usually attributed to the intervertebral disk. It is hoped that this diagnostic procedure may be useful in determining the differential diagnosis and etiology of the symptoms of the so-called whiplash injury, brachial neuritis and cervical cord lesions such as primary lateral sclerosis, multiple sclerosis, cervical cord tumor, etc., all of which may simulate cervical intervertebral disk protrusions.

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SYMMETRICAL FORWARD LUXATION OF THE ATLAS

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DISLOCATIONS of the spine at the atlanto-axial level without associated fracture may be classified on an anatomic basis, as follows: (1) Rotary dislocation of the atlas on the axis (Corner[1]), and (2) symmetrical forward luxation of the atlas on the axis (Coutts[2]).

The etiology[#] of these dislocations has been attributed to trauma (Jefferson[3]) or to infection (Jones[4]).

The present communication is concerned only with symmetrical forward luxation of the atlas. Eleven cases have have been seen by the authors. A further case listed as doubtful is included. A summary of all cases is given in Table 1. An experiment will be described which was designed to prove the roentgenographic appearance of the condition.

Interest was first aroused by the roentgenographic image of the cervical spine of a fifty-five year old man (Case 1, A.L.).

FIG. 1. Case 1. Forward luxation of atlas on axis. L.A.S. 40 per cent.*

* Unless otherwise indicated, the following symbols apply to all figures: A—anterior arch of atlas; P— posterior arch of atlas; O—odontoid process of axis; L.A.S.— loss of available space at the level of the atlas for accommodation of the cervical cord.

REPORT OF A CASE

CASE 1. A.L. The patient had fallen into the cellar of a house on which he had been working as a carpenter. He struck the top of his head, cutting the scalp. He complained immediately of pain in the suboccipital region. Roentgenograms of the cervical spine revealed a forward displacement of the atlas in relation to the axis, thus narrowing the upper cervical spinal canal between the posterior arch of the atlas and the odontoid process (Fig. 1). The anteroposterior views showed no evidence of fracture of the odontoid process. The odontoid was symmetrical in position between the two lateral masses of the atlas.

The patient was treated by ice tong traction for ten weeks. Flexion and extension roentgenograms at that time showed the dislocation to persist. Re-examination six months, eighteen months and finally forty-five months later showed the dislocation still present and to be of as great a degree as originally (Fig. 2, *a* and *b*). At no time did the patient present clinical evidence of compression of the spinal cord at the level of the dislocation. He was asymptomatic

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# See addendum at conclusion of paper.

TABLE I

Case No.	Age	Sex	Symptoms	Neurologic Signs	Per Cent Loss Available Space C1	Associated Congenital Anomalies	History and Type of Injury	Treatment
I. (A.L.)	55	M	Suboccipital pain	None	40	Bifid arch of atlas	Struck top of head during a fall into basement of house	Temporary support
II. (J.M.)	31	M	Occipital pain and pain down left arm. Paresthesias of both hands	Increased reflexes of left upper extremity. Positive Hoffmann's sign of left side	46.5	Extra pair of ribs	None	Fusion neural arches C1-C2
III. (Q.F.)	9	M	Occipital pain	None	25	None	Struck on back of head with piece of ice	Fusion neural arches C1-C2
IV. (B.F.)	76	M	Upper cervical pain	None	37.5	None	Struck on head with heavy rubberized carpet	Temporary support
V. (C.M.)	4	F	Occipital and cervical pain	None (Old brachial plexus palsy)	18	None	Automobile accident. Exact mechanism uncertain	Temporary support
VI. (F.P.)	43	M	Numbness of right hand. Eighteen months later numbness and tingling in hand and feet. Staggering	Bilateral pyramidal tract involvement. Patient dull and apathetic. Fine nystagmus	22	None	Automobile accident. Exact mechanism uncertain	None
VII. (F.S.)	53	M	Momentary unconsciousness. Persistent occipital pain	Immediate quadriplegia with complete recovery during next eight mo.	31	Assimilation of atlas	Fell 7 ft. on to floor of an elevator	Fusion neural arches C1-C2
VIII. (G.H.)	14	M	"Kink" in neck	Torticollis only	44	Assimilation of atlas	Leaped over boards of hockey rink. No direct injury to head or cervical spine	None
IX. (E.H.)	13	M	Cervical pain shooting into right arm	Loss biceps jerk on right side	17	Third occipital condyle	Twisted neck while playing rugby	None
X. (D.M.)	36	M	Cervical pain	None until time of death	76	None	Epileptic seizure resulting in death in a few minutes	Intermittent temporary support
XI. (W.K.)	20	M	Numbness and tingling in hands and feet. Unable to move limbs	None	48.5	Bifid arch of C1 and C2. Left half arch C1 underdeveloped. Interspinous ligament between C1 and C2 missing	Forced flexion of neck during rugby practice	Fusion neural arches C1-C2
Specimen					64			
Doubtful Case (C.G.)	14	M	Unconscious	Subarachnoid hemorrhage	15.5	None	Fell running to base during ball game	None

and refused an operation. When last seen he was carrying on as a carpenter.

Experiment. In order to test the validity of this "apparent" dislocation, a fresh human cervical spine was obtained and attached to a plank in such a manner that the weight suspended from the neural arch of C2 was transmitted directly to the transverse ligament of the atlas (Fig. 3). A weight of 8.2 kg. was dropped approximately 2 feet. There was an immediate rupture of the transverse ligament close to its point of attachment to the inner side of one lateral mass of the atlas.

A laminagram, taken immediately after the experiment, showed an identical picture of the relationship between the atlas and the odontoid process of the axis as was seen in the roentgenograms of Case 1 (Fig. 4). Photographs of the specimen taken before and after the experiment demonstrated the relative forward displacement of the atlas in relation to the axis and the impingement on the cervical cord by the odontoid process anteriorly and the posterior arch of the atlas posteriorly (Fig. 5, *a* and *b*). Thus it would seem that our first patient had escaped serious spinal cord injury by the narrowest of margins.

COMMENT

In a lateral roentgenogram of the normal upper cervical spine, the anterior arch of

Fig. 2. Case 1. Laminagrams showing (*a*) flexion and (*b*) extension of the cervical spine forty-five months following initial injury. Partial reduction of dislocation in (*b*).

the atlas is closely applied to the odontoid process, only 1 or 2 mm. separating these structures. This is the most important relationship to observe when looking for dislocation at this site. Flexion and extension roentgenograms of the cervical spine may show the upper aspect of the atlanto-odontoid articulation to open when the chin is depressed. This produces a V-like deformity, the angle of the V being no greater than 2 or 3 degrees (Fig. 6, *a* and *b*). This is easily produced in a specimen by manual flexion of the atlas on the axis before detachment of any ligaments (Fig. 7).

Borderline cases will be encountered in which the atlas appears to be slightly forward in relation to the axis but in which the symptoms are found to be referred to other pathologic changes. This is illustrated in the case of a fourteen year old boy (G.G., Fig. 8) who stubbed his toe and fell while playing baseball. The boy got up, ran to home plate and then collapsed, becoming unconscious. Spinal tap revealed fresh blood in the cerebrospinal fluid and a carotid angiogram demonstrated an aneurysm originating from the left internal carotid artery. This degree of forward displacement of the atlas may be within normal limits

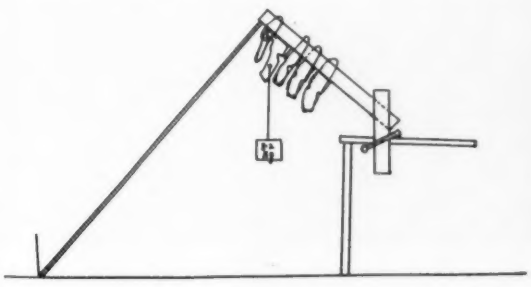

Fig. 3. Apparatus for suspending 8.2 kg. weight from transverse ligament of the atlas.

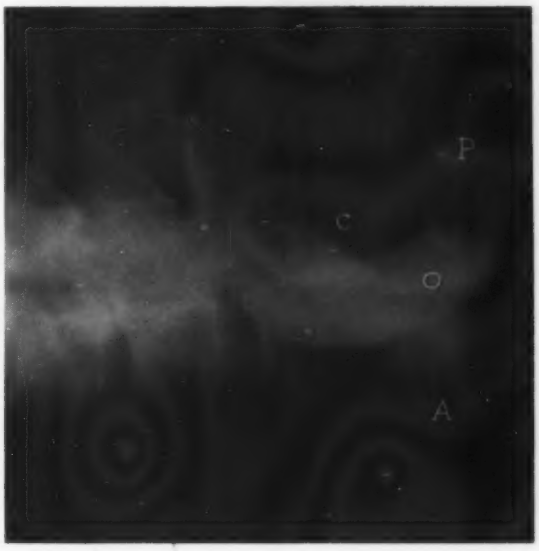

Fig. 4. Laminagram of excised cervical spine used in experiment. C— cervical cord. L.A.S. 64 per cent.,

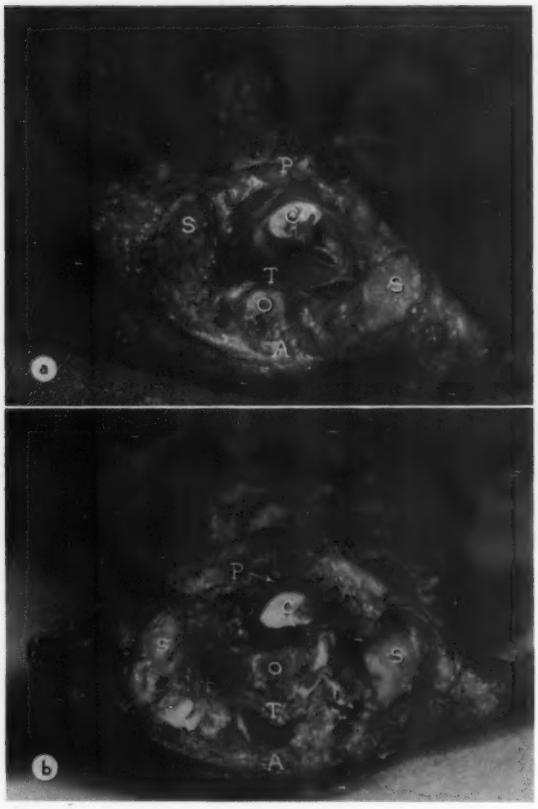

Fig. 5. Excised cervical spine, superior aspect (*a*) before and (*b*) after rupture of transverse ligament of the atlas. S— superior facet of atlas; C— cervical cord; T— transverse ligament of atlas.

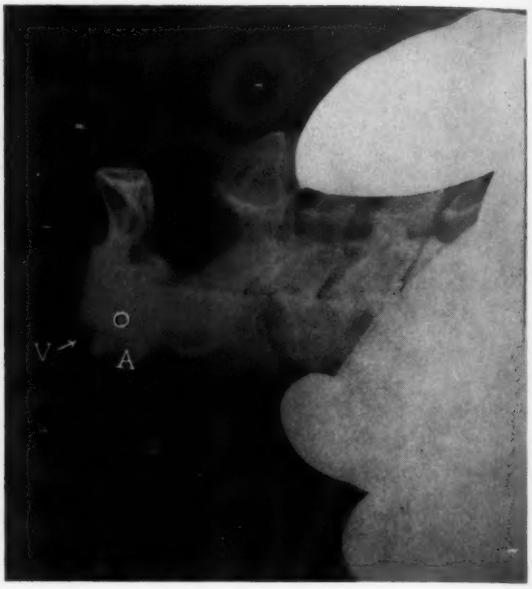

Fig. 7. Excised specimen, manual traction on anterior arch of atlas as in flexion of the spine. Normal V space between atlas and odontoid process of axis.

but it is rarely encountered without an antecedent injury. Reference to the table will show that the actual reduction in the available space for the spinal cord at the level of the atlas in this patient was nearly as great as in some of the other cases which

Fig. 6. Normal atlanto-odontoid articulation. (*a*) Extension and (*b*) flexion of upper cervical spine. Normal V space between atlas and odontoid process when spine is flexed (arrow).

were considered pathologic (Cases v, vi and ix).

Since the first case, 10 additional cases have come to our attention. Histories of the more relevant cases will be given in detail.

REPORT OF ADDITIONAL CASES

Case ii. J.M., male, aged thirty-one, was referred because of pain on the left side of the neck with radiation into the occiput and down the left arm. There was no history to suggest previous infection or serious injury. Physical examination was entirely negative. Roentgenograms demonstrated an extra pair of ribs and, for this reason, it was thought that the complaint might be due to nerve compression at the cervicothoracic level. The patient was discharged and readmitted five months later. At this time there was slight increase in the reflexes of the left upper extremity and a positive Hoffmann's sign on the left side. The patient had paresthesia in both hands. Neurologic opinion on this admission attributed the signs to compression of the cord between the odontoid process and the neural arch of the atlas, the site of the dislocation.

Roentgenograms (Fig. 9, *a*, *b* and *c*) showed forward displacement of the atlas in relation to the odontoid process. In the lateral views, with iodized oil and with air entering the foramen magnum, there was marked reduction in the upper cervical subarachnoid space between the odontoid process and the posterior arch of the atlas (compare with Fig. 10*b*).

Case vi. F.P., male, aged forty-three, was involved in a car accident two years prior to admission. The patient complained of numbness of the right hand, affecting the medial digits. Eighteen months after the accident, the patient developed personality changes, becoming quite easily upset. His face was expressionless. He now complained of numbness and tingling in his hands and feet and began to stagger. He developed a fine nystagmus. On examination, the deep reflexes were hyperactive and Babinski's sign was positive bilaterally. The patient appeared dull and apathetic.

Roentgenograms demonstrated a forward luxation of the atlas on the axis. Air, trapped at the foramen magnum, showed the upper cervical subarachnoid space narrowed at the site of the dislocation (Fig. 10, *a* and *b*).

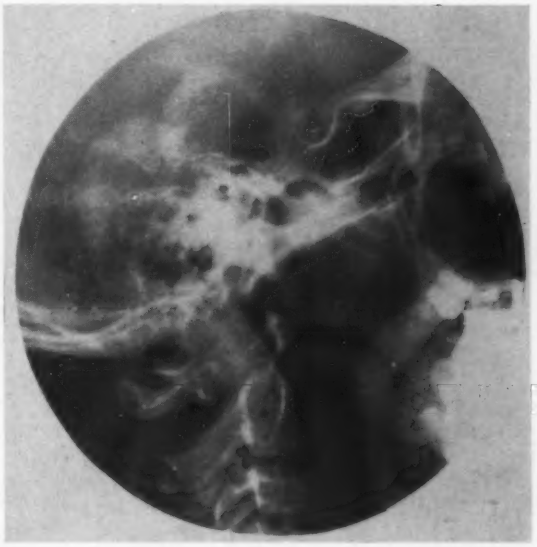

Fig. 8. Borderline case (G.G.). L.A.S. 15.5 per cent.

Case vii. F.S., male aged fifty-three, is an example of forward luxation of an assimilated atlas. This condition has previously been demonstrated by List.[5] The patient fell head first seven feet on to the floor of an elevator when the door gave way suddenly. He was stunned momentarily and, when he regained consciousness, was quadriplegic. After two months in the hospital he was just able to stand up. Eighteen months later his quadriplegia had completely cleared and he attempted to return to work. However, he was unable to do so because of pain in the back of the neck.

Physical examination demonstrated crepitation when the patient turned his head from side to side. There were no neurologic findings but when the patient bent forward he felt dizzy and complained of black spots before his eyes.

Roentgenographic examination (Fig. 11) demonstrated forward displacement of the assimilated atlas. Air at the foramen magnum showed marked reduction of the space between the dorsal aspect of the odontoid and the neural arch of the assimilated atlas.

Case x. D.M., male aged thirty-six, had been a patient in the epileptic wing of the Ontario Hospital, Woodstock, for some years. Epileptic seizures began in 1951. Pulmonary tuberculosis was recognized in 1954. Without an isolated incident resulting in injury to his neck, the patient began to complain of cervical pain in February 1954. In May, 1954 a lateral roent-

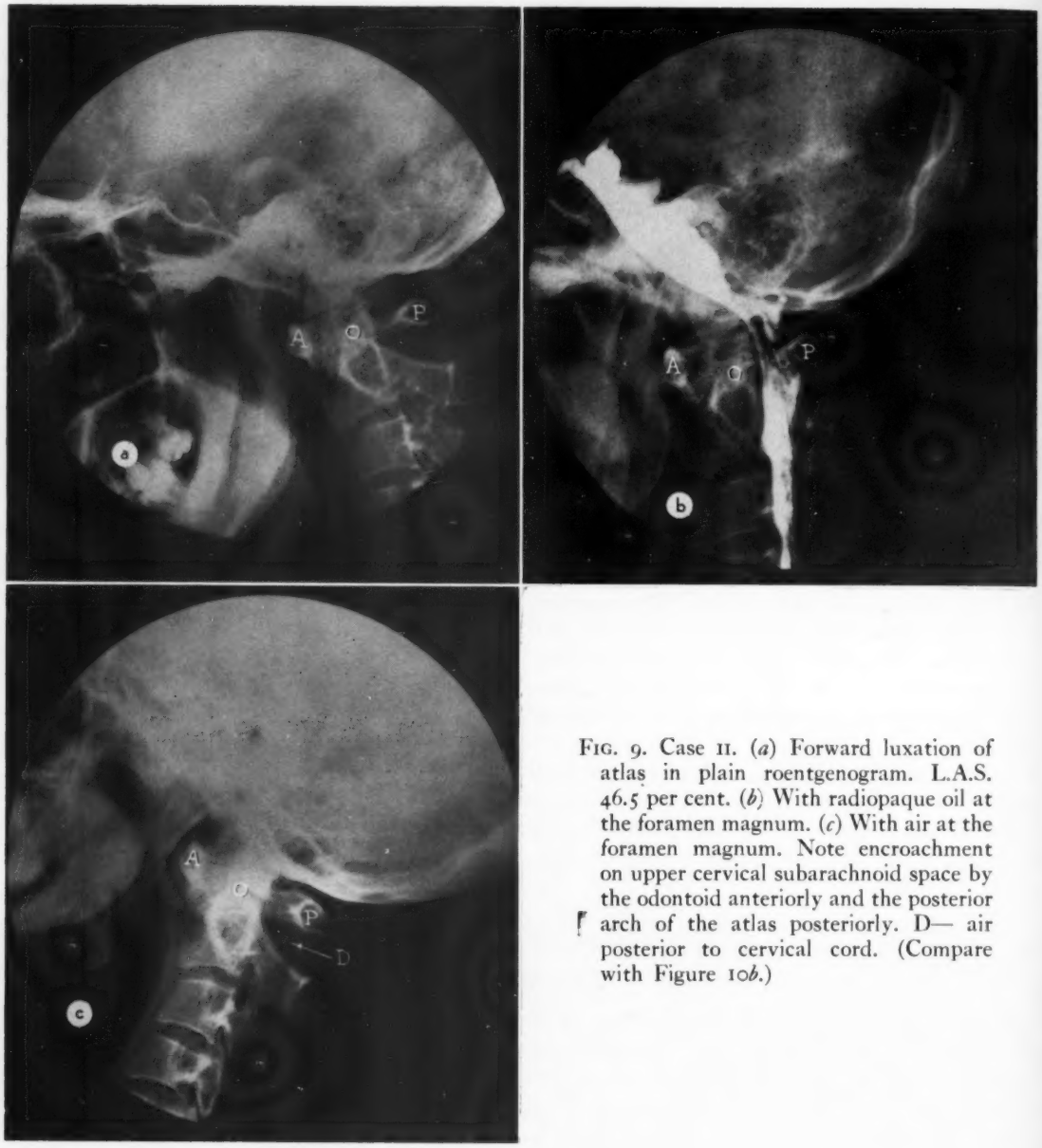

Fig. 9. Case II. (*a*) Forward luxation of atlas in plain roentgenogram. L.A.S. 46.5 per cent. (*b*) With radiopaque oil at the foramen magnum. (*c*) With air at the foramen magnum. Note encroachment on upper cervical subarachnoid space by the odontoid anteriorly and the posterior arch of the atlas posteriorly. D— air posterior to cervical cord. (Compare with Figure 10*b*.)

genogram of the cervical spine appeared normal. In June, 1954 a roentgenogram showed a very marked forward luxation of the atlas on the axis with narrowing of the upper cervical subarachnoid space (Fig. 12*a*). Reduction by extension of the neck and support by means of a collar resulted in a normal relationship between the atlas and the odontoid process (Fig. 12*b*). The support was discontinued after several months. This was followed by an increase in the discomfort and reduction in movement of the cervical spine.

The terminal episode lasted only a few moments. The patient entered the bathroom and was heard to fall heavily to the tile floor, apparently in an epileptic seizure. He received a moderately severe laceration of the scalp. Within two minutes of his fall the patient had expired. Postmortem examination revealed the following: (1) The odontoid process was intact, being neither fractured nor affected by tuberculosis, as was originally suspected; and (2) the spinal cord was softened and disintegrated at the level of C1–C2, having been crushed

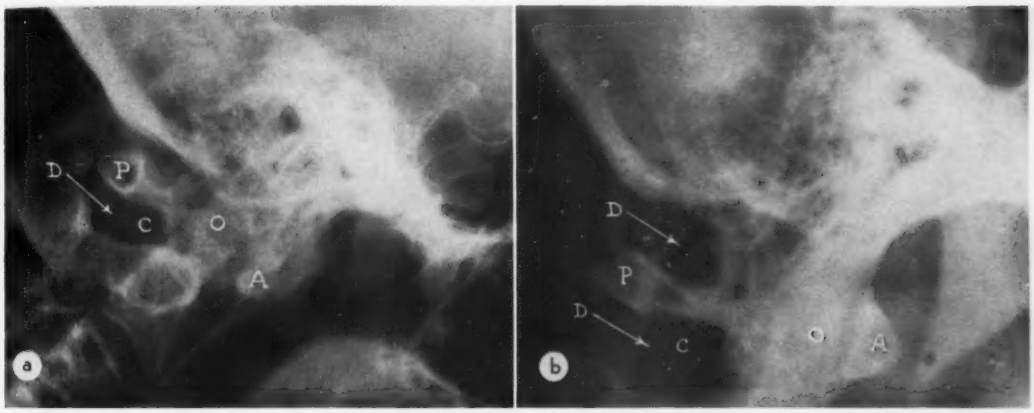

Fig. 10. Case vi. (*a*) Forward luxation of the atlas with air at the foramen magnum. L.A.S. 37.5 per cent. (*b*) Normal cervical subarachnoid space between cervical cord and arch of atlas outlined with air. The cord occupies about a third of the available space. C— cervical cord; D— air posterior to cervical cord and anterior to posterior arch of atlas.

between the odontoid anteriorly and the posterior arch of the atlas posteriorly.

DISCUSSION

Traumatic rupture of a normal transverse ligament of the atlas is described by Jefferson[3] as one of the rarest of all injuries and as one so severe as to result in immediate death. Of Jefferson's reported cases of fracture of the atlas and associated injuries of the upper cervical spine, only 1 suffered rupture of the atlanto-odontoid ligaments without associated fracture. This patient died within a few hours.

Anatomists regard the transverse ligament of the atlas as one of the strongest in the body. To quote Schaeffer[6] in Morris' Human Anatomy, "On its integrity and on that of the alar ligaments our lives largely depend. It is a thick and very strong band, as closely woven as fibrocartilage, about 6 mm. deep at the sides and somewhat more at the midline." Further evidence of the strength of this ligament is born out by the fact that one can suspend a 50 pound weight for twenty minutes on the lateral masses of the atlas with only very slight separation of the atlas and the odontoid.

Fig. 11. Case vii. Forward luxation of assimilated atlas. L.A.S. 31 per cent. D— air posterior to cervical cord.

Histologic sections of a normal transverse ligament, after being stained with special elastic tissue stains, showed no evidence of elastic fibers within the ligament.* In view of these facts, it seems unlikely that the injury in any one of the cases was severe enough to produce rupture of a normal transverse ligament.

No history of infection could be elicited which might be held responsible for weakening this ligament or adjacent soft tissues.

It can be noted in Table 1 that, of the 11 cases, 6 had associated congenital anomalies of the upper cervical vertebrae. This suggested that the cruciate ligament, of which the transverse ligament of the atlas is a

* The authors are indebted to Dr. William Anderson of the Department of Surgical Pathology of the Toronto General Hospital for this observation. Dr. Anderson also obtained the fresh specimens of cervical spine for the experiments.

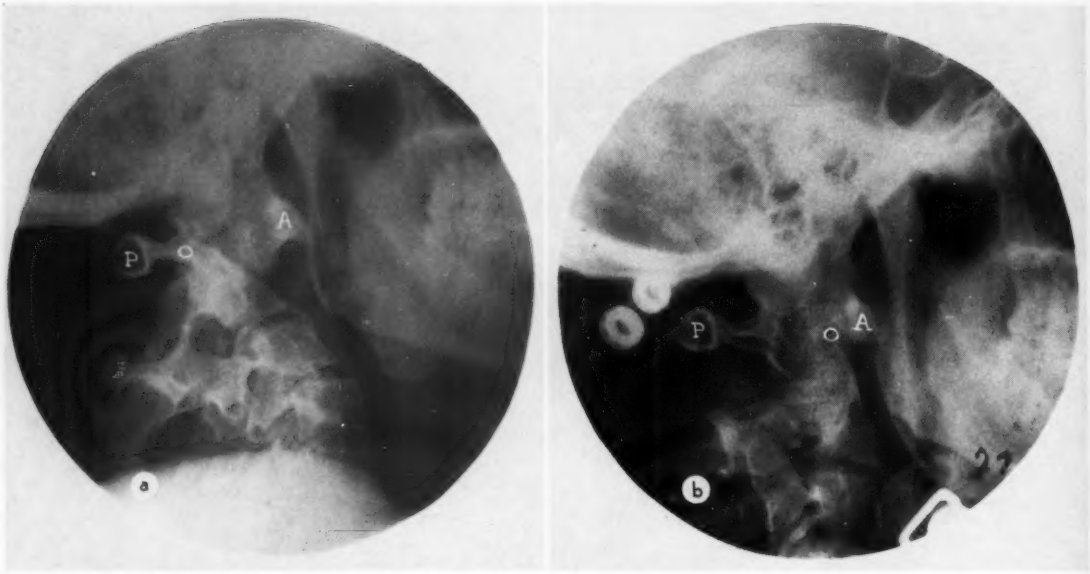

Fig. 12. Case x. (*a*) Gross forward luxation of atlas on axis. L.A.S. 76 per cent. (*b*) Displacement held reduced by use of cervical collar. Death followed epileptic seizure.

part, was congenitally absent or attenuated. McRae[6] describes 6 cases of this nature in which 3 had associated congenital bony abnormalities. This author mentions the possibility of congenital underdevelopment or absence of the cruciate ligament.

Study of Table I demonstrates that there is no correlation between the degree of reduction of the space at the level of the ring of the atlas by this dislocation and the presence of neurologic signs. Thus, Case VI with only 22 per cent reduction later developed signs of cord injury, whereas Cases I and VIII, with 40 per cent and 44 per cent reduction, respectively, had no demonstrable neurologic findings. The laminagram of the specimen showed the available space at the level of the atlas to be reduced by 64 per cent and the photographs demonstrated the odontoid pressing the cord against the neural arch of the atlas. The case considered borderline (G.G.) showed a 15 per cent loss of available space at C1, whereas Case IX, considered abnormal, showed only 17 per cent.

Four cases were followed for an interval of time (Case I—forty-five months, Case II—six months, Case VIII—twenty-four months, Case IX—twenty-four months). In none of these was there evidence of a change in the degree of dislocation during the time observed. Only 4 of the 11 cases were treated by permanent fixation (fusion between the neural arches C1–C2).

SUMMARY

1. Eleven cases of forward luxation of the atlas are discussed. In some, a relatively minor injury accompanied the dislocation; in others, no evidence of injury was elicited. There was no history of infection in any of the cases to account for the dislocation.

2. Six of the 11 cases had associated congenital abnormalities of the cervical spine. This suggested that attenuation or underdevelopment of the cruciate ligament of the atlas accounted for the apparent ease with which this dislocation occurred.

3. The roentgenographic appearance was proved by experiment.

4. Within wide limits there was no correlation between the degree of dislocation and the presence of cord compression.

5. Only 1 death was recorded, but it is believed that this is a potentially serious type of dislocation. The relatively ample

space for the cervical cord at the level of the atlas spared the cervical cord from serious compression in the remaining patients.

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The authors are grateful to the following physicians for permission to publish their private cases: Dr. F. P. Dewar, Toronto (Case III), Dr. N. Laurie, Barrie (Case V), Dr. R. I. Harris, Toronto (Case VII), Dr. E. C. Evans, Physician, Ontario Hospital Service, Woodstock (Case X), Dr. G. W. Kruger, Radiologist, Woodstock General Hospital, Woodstock (Case X), Dr. J. L. Peniston, Pathologist, Stratford General Hospital, Stratford (Case X), Dr. George Pennal, Toronto (Case XI), Dr. Wallace Roy, Toronto (Case XI).

ADDENDUM

Since this manuscript was prepared, 3 additional cases have come to the attention of the authors. These were associated with the following conditions: (1) ankylosing spondylitis; (2) osteoarthritis; and (3) von Recklinghausen's neurofibromatosis. Thus, in considering the etiology of these dislocations, acquired disease of the transverse ligament itself must be added to those already mentioned.

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ROENTGENOLOGIC ASPECTS OF BENIGN CHONDROBLASTOMA OF BONE*

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BENIGN chondroblastoma is a neoplasm of bone which has been recognized as a distinct entity for a relatively short time. In the past it frequently has been mistaken for other neoplasms of bone of more serious consequence, notably chondrosarcoma and giant-cell tumor of bone. This error has resulted in unnecessarily radical surgical procedures with consequent physical and psychologic handicaps, usually in young patients.

REVIEW OF LITERATURE

The first report in the literature, describing the lesion now known as benign chondroblastoma, was by Ewing[6] in 1928. He described the tumor under the name of "calcifying giant cell tumor." He recognized the benign nature of the lesion and also that it was often mistaken for a malignant tumor of the bone.

Codman[3] gave the first detailed description of this tumor in 1931. He reported 9 cases in which tumors occurred in the upper end of the humerus. He had collected these cases from the Registry of Bone Sarcoma of the American College of Surgeons and reported them under the title of "Epiphyseal Chondromatous Giant Cell Tumors of the Upper End of the Humerus" because he held the opinion that they were a clinical entity confined to the upper end of the humerus but that they were essentially giant-cell tumors.

In 1942, Jaffe and Lichtenstein[13] proposed the name "benign chondroblastoma" for a group of 9 tumors which had originally been classified in their files as "variants" of giant-cell tumor. They considered benign chondroblastoma to be a primary tumor of bone which developed from cells best interpreted as cartilage germ cells, and that this tumor was quite distinct from the ordinary giant-cell tumor.

Geschickter and Copeland[9] stated that these tumors may be either benign or malignant and in one study they listed 10 tumors as benign and 16 as malignant chondroblastic tumors.

No other authors in the literature have published reports of malignant degeneration of previously benign tumors of this nature and no others have called chondroblastoma a primary malignant neoplasm. However, various authors have reported tumors as malignant neoplasms which, on review, appear to have been benign chondroblastomas.

At the time that this paper was written (April, 1957) the literature contained reports of 74 cases in which tumors were diagnosed as benign chondroblastoma or epiphyseal chondromatous giant-cell tumor.[1,2 4,5,7,8,10–12,14–20] Four other tumors have been found under different diagnoses which we believe should be classified as benign chondroblastoma and would make a total of 78 cases.

MATERIAL AND METHODS

The files of the Mayo Clinic showed a diagnosis of benign chondroblastoma in 17 cases. Fifteen of these were pure benign chondroblastomas. Two tumors initially contained elements of both chondroblastoma and chondromyxoid fibroma. One of

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† The Mayo Foundation, Rochester, Minnesota, is a part of the Graduate School of the University of Minnesota.

these recurred and microscopically was typical chondromyxoid fibroma of bone. The clinical records of the 17 cases were reviewed and the gross and microscopic pathologic factors were studied. Roentgenograms were available in 10 cases and photographs of roentgenograms in 4 cases.

CLINICAL DATA

The age of the patients affected with this tumor ranged from eight to fifty-nine years. Two patients were in the first decade of life, 8 in the second, 2 in the third, 1 in the fourth, 1 in the fifth, and 3 in the sixth. Of the 17 patients, 10 were males and 7 were females.

The distal femur was involved in 4 patients, the proximal femur in 3, and the proximal portion of the humerus in 3. The scapula was affected in 2 patients and the bones of the pelvis in 4. A rib was the site of one of these tumors. It will be noted that the site of involvement was the long bones of the extremities in 10 cases and the flat bones of the pelvis, scapula, and rib in 7 cases.

Pain was the most common symptom; it occurred in 16 patients. Tumor or swelling was noted by 7 patients and 6 complained of limp. Two patients complained of weakness and stiffness of the affected extremity. Limited motion, numbness, and loss of weight were each noted once.

A history of trauma was present in 4 instances but did not appear to have any significant relationship to the development of the tumor.

Physical findings included tumor or joint swelling, limited or painful motion, tenderness or pain on palpation, and muscular atrophy.

PATHOLOGIC ASPECTS

Gross Appearance. Four resected specimens, in which the entire tumor was available, were studied. In all 4 specimens the neoplastic tissue was sharply demarcated from the surrounding normal bone by a thin margin of sclerotic bone which was either smooth or slightly ridged but was without gross bony trabeculation.

One tumor which involved the innominate bone bulged into the pelvic cavity as a smooth, ovoid mass. It was covered by a very thin shell of bone that was unbroken and well limited by periosteum.

In none of the resected specimens did neoplastic tissue infiltrate the soft tissues adjacent to it. The tissue varied considerably in appearance. Some specimens consisted almost entirely of gray or bluish-white cartilage-like material; others were yellowish brown. Consistency of the tissue varied from friable to moderately firm. Gross calcification or ossification within the neoplastic tissue was present in only 3 tumors.

Microscopic Findings. The basic stromal cell of these tumors was a round, oval, or polyhedral cell with a relatively large round or oval nucleus. The cells were usually rather compact with little intercellular connective tissue. Scattered throughout the stromal cells were irregular islands of chondroid tissue which varied greatly in size and which stained either eosinophilic or slightly basophilic with hematoxylin and eosin. Within this ground substance there were viable cells which were round, polyhedral, or multipolar; sometimes they appeared to be contained in lacunae. Some of the chondroid islands were sharply demarcated from the stromal cells while others merged gradually with them.

Multinucleated giant cells were present in variable numbers interspersed throughout the stromal cells.

Small areas of osteoid and bone were present in many of these tumors. Sometimes they merged imperceptibly with islands of chondroid substance suggesting transformation of chondroid into osteoid and bone.

Microscopic evidence of calcification was found to be present to a variable degree in 12 of these tumors. It was present to a greater extent within the chondroid substance than elsewhere. Occasionally calcification was present in the stromal cells and in small islands of osteoid.

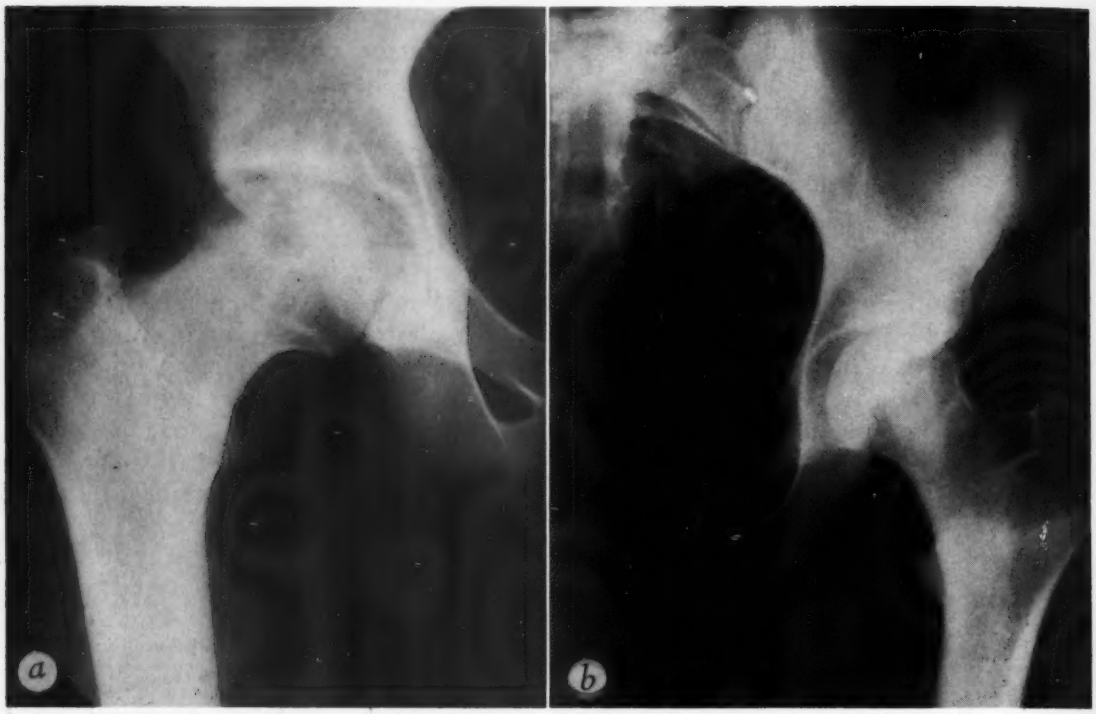

Fig. 1. (*a*) Well-circumscribed benign chondroblastoma located in the head of the femur. Note the thin margin of sclerosis of bone surrounding the tumor. (*b*) Benign chondroblastoma involving the greater trochanter and neck of the femur. Slight mottling is evident within the area of rarefaction. The tumor is sharply limited from normal bone by a thin line of sclerosis.

One of the tumors in the distal femur of a ten year old boy contained typical benign chondroblastoma and also regions of oval, spindle-shaped, or multipolar nuclei within a myxoid intercellular ground substance which is characteristic of chondromyxoid fibroma of bone. Another tumor in the distal femur of an eight year old boy originally contained a mixture of benign chondroblastoma and chondromyxoid fibroma but recurred as a pure chondromyxoid fibroma nine years after curettage and roentgen irradiation.

TREATMENT AND PROGNOSIS

Two patients were treated by amputation and two by resection; one had biopsy followed by roentgen irradiation and one had biopsy alone. The remaining 11 patients had curettage or local excision and 6 of these received postoperative roentgen therapy.

One tumor recurred nine years after curettage and roentgen therapy; follow-up study showed the patient to be well nine years after the second treatment. One tumor in the pelvis recurred ten years after hindquarter amputation. The patient was found to be well sixteen months after the second treatment. There was no follow-up on one patient. The remaining patients are living and well from two and one-half to twenty-one years after treatment.

ROENTGENOLOGIC FINDINGS

Roentgenograms of 10 and photographs of roentgenograms of 4 tumors were available for study. In 8 roentgenograms lesions were evident in the long bones. Three lesions were confined to the epiphysis; 1 was confined to the metaphysis but abutted on the epiphyseal cartilage plate. The remaining 4 tumors in long bones involved the epiphysis and adjacent metaphysis. In the 2 cases involving long bones in which roentgenograms were not available the

surgeon's notes stated that the lesions involved both epiphysis and adjacent metaphysis. Lesions that extended into the metaphysis usually were eccentrically located.

All flat bones which contained lesions were bones which are preformed in cartilage.

The size of the tumors varied from 1.5 by 1.8 cm. to 8.0 by 19.0 cm. The average size was about 5.0 by 7.0 cm.

The tumors appeared oval in all instances in which margins of the tumor could be seen (Fig. 1, *a* and *b*, and 2). Lesions which extended completely to the cortex either conformed to the shape of the cortical margin or caused an ovoid expansion of the cortex. The margin of the tumor which was limited within the medullary cavity was usually curved. Slight scalloped irregularity of contour occurred occasionally.

FIG. 3. Large expanding lesion in the pelvis caused by benign chondroblastoma originating from the innominate bone. Note the mottling within the center of the tumor and the sharply defined sclerotic margins.

FIG. 2. Expanding benign chondroblastoma in the wing of the right ilium. Note the mottled appearance due to calcification and ossification within the tumor and also the marked sclerosis at the margins of the tumor.

A very characteristic roentgenographic finding of all these tumors was a thin sharply defined margin of sclerotic bone which separated the tumor from the adjacent spongiosa (Fig. 1, *a* and *b*, and 2 and 3). This line was not completely visualized in the tumor which involved the proximal humeral epiphysis (Fig. 4). It was partially obscured by thickening of adjacent uninvolved bony trabeculae which had the appearance of "densified bone beyond the limits of the tumor" described by Jaffe and Lichtenstein. However, gross examination of the resected specimen revealed that the tumor was well limited from the surrounding bone by a thin sclerotic margin of bone. In the roentgenograms of a tumor which affected the ischium and ilium, the margin of the tumor in the ischium adjacent to the pubis lacked the thin, sharp delimiting line, but the junction between tumor and normal bone was quite distinct (Fig. 5*a*).

The mottled, fuzzy, fluffy cotton-wool appearance which has been described in the literature as being characteristic of benign chondroblastoma was present in 8 and absent in 6 roentgenograms (Fig. 1*b*, 2, 3 and

Fig. 4. Benign chondroblastoma of the proximal portion of the humerus. Biopsy had been carried out previously, but the appearance of the tumor in this projection was not altered. There are thickening of the cortex medial to the tumor and increased density of the spongiosa distal to it.

4). There was close correlation between lack of mottled appearance on the roentgenogram and absence of microscopic evidence of calcification.

The dense conglomerate calcification often seen in enchondroma or chondrosarcoma was not evident in any roentgenograms of these lesions prior to treatment. It was seen in a large lesion involving the scapula eleven years after biopsy and roentgen therapy.

Roentgenographic evidence of expansion of the involved bone was present in 10 tumors in this series. Marked thinning or complete destruction of the overlying cortex with a soft-tissue mass was caused by a few of these tumors (Fig. 5*b*). This destruction appeared to be due to gradually increasing expansion and not to active invasion of bone as is seen with a malignant neoplasm. There was no evidence of irregular soft-tissue invasion or production of bone in adjacent soft tissue.

Evidence of active periosteal reaction was seen in only 1 roentgenogram of this group. The reaction in this case occurred

Fig. 5.(*a*) Benign chondroblastoma expanding the ilium and ischium. The extent of the large soft-tissue mass extending into the pelvic cavity is shown by the arrows. This tumor might easily be mistaken for a malignant neoplasm. (*b*) Benign chondroblastoma causing marked expansion of the scapula. Observe the large, well-defined, soft-tissue mass, the sclerotic margins, and the appearance of slight trabeculation within the tumor.

medial to the neck of the humerus. Other evidence of periosteal reaction appeared as cortical thickening in 4 instances. The so-called densified bone beyond the limits of the tumor described by Jaffe and Lichtenstein was observed 4 times (Fig. 4).

Trabeculation, somewhat similar to that seen in some giant-cell tumors or aneurysmal bone cysts, was present in only 1 tumor; this was one which greatly expanded the scapula (Fig. 5*b*). Very slight irregular trabeculation was noted in 2 other lesions.

Part of the articular cortex was destroyed by one tumor located in the capital femoral epiphysis.

DIFFERENTIAL DIAGNOSIS

In the differential diagnosis of benign chondroblastoma from the roentgenologic viewpoint, enchondroma and chondrosarcoma should be considered carefully. Enchondroma and rarely chondrosarcoma may involve the epiphysis and adjacent metaphysis and be fairly well demarcated from normal bone by a thin margin of sclerotic bone. If calcification within a tumor is conspicuous roentgenographically, the lesion is probably either an enchondroma or a chondrosarcoma rather than a benign chondroblastoma. Mottled, indefinite calcification also may be present in enchondroma or chondrosarcoma; in this case roentgenographic differentiation from chondroblastoma is impossible if there is a well-defined line of marginal sclerosis surrounding the tumor and if the epiphysis and adjacent metaphysis are involved.

The lesions of fibrous dysplasia or nonosteogenic fibroma of bone are sometimes delimited from the surrounding bone similar to benign chondroblastoma. However, they rarely involve the epiphysis and often appear trabeculated with more scalloped margins than those caused by benign chondroblastoma. Multiplicity of lesions involving the metaphysis and diaphysis is often seen in fibrous dysplasia but has never been reported due to chondroblastoma. Solitary lesions of fibrous dysplasia or nonosteogenic fibroma involving the flat bones of the pelvis or scapula may pose a more difficult problem in differential diagnosis; in fact, differentiation may be impossible roentgenographically.

Giant-cell tumor involving the long bones must be considered in the differential diagnosis of chondroblastoma. However, if the lesion has not been treated previously by curettage or roentgen irradiation or both, the differentiation should not be difficult. The giant-cell tumor, like chondroblastoma, almost always affects the epiphysis of the long bone; but if the adjacent shaft is involved, the untreated giant-cell tumor, unlike most chondroblastomas, does not have the sharply defined sclerotic margin dividing it from the normal medullary bone. The giant-cell tumor does not have the mottled appearance owing to calcification which may be seen in some cases of benign chondroblastoma. Giant-cell tumor affecting the flat bones should not be confused with chondroblastoma. It almost always has irregular margins without surrounding sclerosis and must be differentiated from solitary myeloma or metastasis rather than chondroblastoma.

Chondromyxoid fibroma does not appear to involve the epiphysis as often as does benign chondroblastoma, and it usually does not have the mottled appearance that some chondroblastomas have owing to calcification.

If chondromyxoid fibroma involves the epiphysis and adjacent shaft, it may be impossible to distinguish between it and benign chondroblastoma on the basis of roentgenographic appearance.

Eosinophilic granuloma of bone, in its active phase, usually has irregular, ill-defined margins and does not offer any problem in differential diagnosis. However, healing lesions may be surrounded by a thin line of sclerosis, especially after irradiation or surgical curettage; in such cases, solitary lesions involving the flat bones may be difficult to distinguish from chondroblastoma roentgenographically. Multiplicity of lesions is often a characteristic of eosinophilic

granuloma, and if other lesions are found their presence is suggestive evidence that the lesion in question is not benign chondroblastoma.

Giant-cell tumor and myeloma may develop a margin of sclerotic bone after roentgen treatment or surgical curettage; this also may occur in treated metastatic carcinoma such as metastatic carcinoma from the breast treated by irradiation or hormonal therapy or both. Such lesions involving flat bones may pose a problem in differential diagnosis and it is therefore important that the roentgenologist be informed of previous therapy.

Inflammatory diseases of bone which are chronic and slowly progressive or arrested may produce a thin margin of dense bone at the periphery of an area of destruction and produce a lesion roentgenographically indistinguishable from benign chondroblastoma. Only 1 such case was found among the cases encountered at the Mayo Clinic. In this case the lesion occurred in the humerus and involved the proximal epiphysis and adjacent metaphysis; it was caused by brucellosis which had been present for several years.

One case of capillary hemangioma located in the head and neck of the humerus was found which, from a roentgenologic viewpoint, could not be differentiated from chondroblastoma. Capillary hemangioma involving bone is a rare condition and the pathologist will not have difficulty in differentiating it from benign chondroblastoma.

SUMMARY AND CONCLUSIONS

Benign chondroblastoma is a neoplasm of bone of cartilaginous origin. No authenticated malignant transformation of a previously benign lesion has been reported.

All but 6 tumors reported in the literature as benign chondroblastoma have involved the long bones; 5 of the 6 affected bones of the hands or feet and 1 involved the temporal bone. A giant-cell tumor in the sternum reported in one case[18] seemed, on further study, to be a chondroblastoma. At the Mayo Clinic 17 cases of chondroblastoma were encountered; 7 tumors involved the pelvis, scapula, and rib, sites not previously reported in the literature.

The most characteristic roentgenographic feature of this neoplasm is a central area of destruction of bone which is sharply limited from the surrounding normal bone, usually by a thin margin of increased density of bone. There may or may not be an appearance of mottling within the radiolucent area. Both trabeculation and active periosteal reaction are rarely seen. The tumor, when it involves the long bones, almost always affects the epiphysis and frequently the adjacent shaft. Rarely the shaft alone will be involved and then the tumor abuts onto the epiphyseal plate. We have not found a lesion of this type in long tubular bones involving only the diaphysis at a distance from the epiphyseal plate in this series and none has been reported in the literature.

A smooth, well-outlined soft-tissue mass may be seen in large tumors which have destroyed the overlying cortical bone by pressure erosion.

The most important lesions which should be considered in the differential diagnosis roentgenographically and pathologically are enchondroma, chondromyxoid fibroma (which seems to be closely related to benign chondroblastoma), chondrosarcoma, and giant-cell tumor. Rarely an inflammatory lesion or a solitary focus of fibrous dysplasia may simulate it roentgenographically. Metastatic carcinoma, solitary myeloma and solitary lesions of eosinophilic granuloma may resemble it after roentgen irradiation or curettage, especially in flat bones. Therefore, it is essential that the roentgenologist be informed of any previous diagnosis and therapy before rendering an opinion about such a lesion. Also, close cooperation between the radiologist, pathologist, and surgeon is urged to facilitate correct diagnosis of this tumor and thus prevent unnecessarily radical surgical procedures.

The treatment of choice is surgical curettage.

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JUXTACORTICAL OSTEOGENIC SARCOMA*

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BECAUSE certain bone tumors are infrequently seen, they present problems in precise diagnosis and treatment. A lesion of this variety is herein described which has not previously been reported in the American radiologic literature. It has been variously named periosteal osteogenic sarcoma, parosteal osteoma, juxtacortical osteogenic sarcoma, and parosteal osteogenic sarcoma. The recognition is far from academic for instead of the 10 per cent survival in osteogenic sarcomas as a group juxtacortical osteogenic sarcoma when properly treated may well have a five year survival in excess of 90 per cent.

We shall attempt to advance viewpoints for diagnostic and therapeutic criteria. These concepts are not presented with the claim of originality but rather reflect current concepts many of which have been suggested by Jaffe. However, the authors take the responsibility for the conclusions presented.

We prefer the term juxtacortical osteogenic sarcoma in reference to an ossifying lesion which occurs on the surface of the long bones, the lower end of the femur being a favorite site of predilection. The lesion is asymptomatic and is unnoticed until pressure effects are developed by the patient. If detected early the lesion may be successfully treated by a complete local excision; however, if inadequately removed it will recur and is capable of metastasizing, usually to the lungs. If the lesion cannot be completely eradicated by local excision amputation is recommended with an adequate amount of uninvolved tissue. Usually, because of their insidious onset, juxtacortical lesions of this type have advanced to a point where amputation remains as the only safe choice for a cure.

REPORT OF A CASE

This eighteen year old white male was admitted on October 14, 1956, with the chief complaint of a mass present for two years above and behind the left knee which interfered with complete flexion. For two months prior to admission he had experienced night pain and a vague sense of discomfort in the left knee. Physical examination revealed a firm nodular 6 by 8 cm. mass on the posterior supracondylar aspect of the left femur. This mass was slightly tender but there was no local heat over the area and a loss of only some 10 degrees of flexion at the knee was noted. The remainder of the physical examination was unremarkable.

Roentgenograms revealed a 6 by 8 cm. calcified mass with its broad base attached to the posterior supracondylar region of the left femur. The periphery was lobulated and scattered throughout the calcified tumor were small radiolucent areas suggesting a cartilaginous component. There was evidence of periosteal new bone formation adjacent to the mass and Codman's triangle was, therefore, a prominent part of the picture. By exposures in various projections and laminagraphy the underlying cortex was demonstrated to be widened but no destruction or breakthrough was noted. The medullary canal was not involved roentgenographically. The surrounding soft tissues showed extrinsic pressure involvement only. A preoperative diagnosis of osteogenic sarcoma of the juxtacortical type was made (Fig. 1, 2 and 3).

At the time of open biopsy the posteroinferior portion of the mass appeared to be primarily bone intermixed with friable cartilaginous tissue. The microscopic examination revealed areas of fibroblastic proliferation with plump spindle cells; no mitotic figures were

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The opinions expressed herein are those of the authors and do not necessarily represent the views of the Bureau of Medicine and Surgery, Navy Department.

Fig. 1. Anteroposterior roentgenogram of the distal one-third of the left femur.

Fig. 2. Soft tissue reontgenogram of this same area.

Fig. 3. Lateral roentgenogram of the distal left femur. Laminagraphy confirmed the impression of an intact medulla suggested by this film.

seen (Fig. 4). The histopathologic diagnosis was juxtacortical osteogenic sarcoma.

A week after the biopsy a midthigh amputation was performed. Pathologic examination of the resected specimen revealed a palpable firm tumor in the area of the knee. There was a recent 7 cm. surgical skin incision in this area. When the soft tissues overlying the tumor were removed, a hard lobulated bone tumor was visualized, intimately adherent to the cortex posteriorly and laterally (Fig. 5). This mass extended from the intertrochanteric notch upward for a distance of 8 cm. It was 7 cm. at its widest portion. On cut surface (Fig. 6) the tumor was continuous with the posterior cortex of the femur and no extension into the medulla was noted. The tumor tissue contained areas of cartilaginous, fibrous, and osseous matrix. Microscopic examination of the tumor revealed closely packed spindle cells, cartilage and bone spicules (Fig. 7). In some areas the spindle cells were moderately pleomorphic with plump nuclei and prominent nucleoli (Fig. 8). In no section was there involvement of the medulla.

FIG. 4. Photomicrograph of the biopsy specimen (250×).

FIG. 5. Gross appearance of the tumor, posterior view.

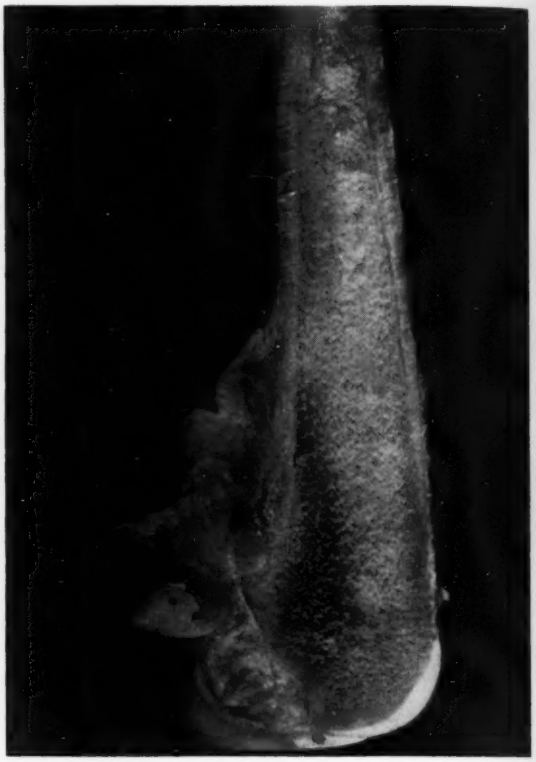

FIG. 6. Appearance of the cut surface of the tumor. The dark portion of the medullary canal immediately beneath the cortex adjacent to the tumor represents hyperemia only.

The final diagnosis was juxtacortical osteogenic sarcoma of the left femur.

HISTORICAL REVIEW

Although this unique type of osteogenic sarcoma was mentioned briefly by Coley[2] in 1951, it remained for Geschickter and Copeland[4] in that same year to recognize the necessity for a specific label. They collected 16 cases and coined the phrase "parosteal osteoma" which was somewhat misleading since of their cases 13 were proved malignant. In 1952, Jaffe and Selin[6] in a symposium spoke of the favorable prognosis as compared to the conventional intramedullary type of sarcoma.

Dwinnell, Dahlin, and Ghormley[3] in 1954 finally put the present day label of parosteal (juxtacortical) osteogenic sarcoma on the entity to indicate the true nature of the disease. They presented 15 cases obtained

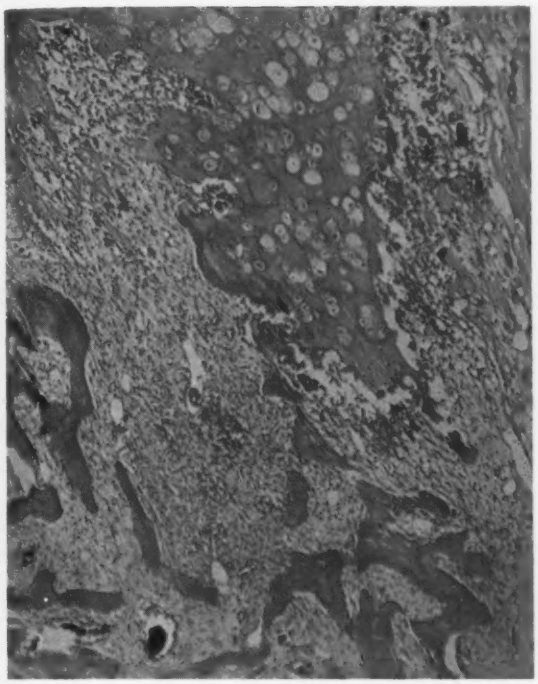

Fig. 7. Photomicrograph of the tumor, low power (100×).

Fig. 8. Photomicrograph of the spindle cell area (200×).

by reviewing some 2,000 primary bone tumors including some 400 osteogenic sarcomas. They further supported the exclusion of heterotopic ossification as a source for this lesion. Since that time Lichtenstein[7,8] has briefly mentioned this peculiar tumor in articles covering groups of bone tumors.

INCIDENCE AND DISTRIBUTION

Because of the small number of reported cases, significant statistics are inconclusive. However, it appears that these tumors will account for approximately 0.8 per cent of all primary bone tumors and for less than 4 per cent of osteogenic sarcomas. There appears to be no significant sex difference and the age range extends from ten to fifty years with a predominance in the third and fourth decade.

The site of predilection for 70 per cent is the distal posterior aspect of the femur. The upper humerus is responsible for most of the remainder with the upper fibula and ulna reportedly having been involved.

It is likely that the 1939 Bone Tumor Registry's classification of osteogenic sarcoma contains cases of juxtacortical osteogenic sarcoma under the subcategory of capsular and periosteal. These tumors may also have masqueraded under designations such as "atypical osteochondromas" or "strange recurring forms of myositis ossificans."

CLINICAL ASPECTS

The symptomatology of these patients is remarkably similar in that most have noted a mass for months or years and experienced little or no discomfort. Fever, pain, anemia, weight loss, leukocytosis, and other symptoms sometimes associated with malignant bone tumors are absent. Physical examination usually reveals a large, firm, nontender, nonmovable mass in the region of the metaphysis or epiphysis of a long bone. There is limitation of motion only because of the mechanically strategic location of the tumor. Local heat is noted in less than one-third of the cases.

ROENTGEN FINDINGS

The initial lesion, attached to the metaphyseal or adjoining epiphyseal region, is a lobulated juxtacortical ossifying mass with scattered areas of lesser density representing cartilage, trapped fascia, and muscle. The underlying cortex, encroached upon by the broad-based mass, becomes irregular and thickened; but in view of the density and size of the tumor there is surprisingly little reaction or destruction. In the adjacent periosteum a small Codman's triangle (the zone of reactive bone formation adjacent to tumor periosteum) is often seen. Tumor growth is along the shaft and circumferentially sometimes to the extent of surrounding the bone with the shaft coursing through the tumor. When first examined roentgenographically at this advanced stage the erroneous impression of medullary involvement may be suggested; right angle laminagraphy and stereoscopy may be of help in excluding cortical invasion. The periphery of the tumor may blend somewhat with denser soft tissue structures, but as a rule it is rather sharply demarcated.

After an inadequate local excision an irregular cortex is noted which soon shows renewed activity with dense irregular osseous tumor invading soft tissue. Eventually, often after two or more unsuccessful attempts at local excision, frank sarcomatous manifestations consisting of combined sclerosis and osteolysis with destruction of the cortex and medullary cavity become evident.

This unique and characteristic roentgenographic appearance, especially coupled with a history of a relatively asymptomatic mass of long standing, in a young person indicates strongly the possibility of a juxtacortical osteogenic sarcoma.

DIFFERENTIAL DIAGNOSIS

1. Osteogenic sarcoma. This is differentiated by local pain, systemic toxicity, rapid growth with almost universal medullary involvement and osteolysis of cortex with or without osteosclerosis and "sunray" reaction produced by the periosteum.

2. Osteochondromas and exostoses. The bases of these tumors are continuous with the cortex and medullary canal of the shaft. The trabecular pattern within the tumor is orderly and there are no areas of radiolucency scattered throughout the mass since fascia and muscle are not contained therein and the cartilage forms a cap. Malignant change is rare, but when it does occur the roentgenogram fails to reveal any osseous component as the metamorphosis is to a pure chondrosarcoma. These differential points also apply to periosteal chondromas.

3. Sesamoidomas. These are actually intraligamentous osteomas and are smaller, discrete, sharply defined, and fail to show progressive growth. Roentgenograms with a soft tissue technique can be of assistance by demonstrating the tumor to be localized to a tendon sheath or ligament.

4. Heterotopic ossification. This is associated with a history of trauma in two-thirds of the cases. The swelling reaches a maximum in one to three months and then remains unchanged. The roentgenogram reveals dense laminated ossification paralleling the shaft and following the fascial planes of the muscle. Underlying cortical thickening and periosteal reaction is absent as expected since by proper projection or laminagraphy the mass and underlying bone can often be separated.

5. Differentiation of the reparative phase following minimal fractures, especially in osteogenesis imperfecta, may be difficult from the initial roentgenogram, but history and histologic examination render the diagnosis evident.

PATHOLOGY

The tumor arises from the periosteum and then retains bone forming tendencies; on gross examination the islands of calcification are prominent; histologically, the interstices between the bone spicules are filled by spindle cell fibroblasts; the nuclear variability is a function of the activity of the tumor and bizarre forms may be seen

which herald a malignant tendency. The clinical history, roentgenographic appearance, and multiple histologic sections must all be inculcated in attaining an accurate portrayal of the pathologic implication of this tumor.

THERAPY

Several authors,[3,4] believing that all lesions of parosteal ossification represent low grade sarcomas from their inception, recommend amputation even in the absence of unequivocal histologic evidence of malignant change. Lichtenstein[7,9] believes this to be an unjustified, rather extreme point of view. He states, however, that the pathologist must be on the alert for this serious complication and must sample all of the tissue submitted, scrutinizing especially closely the fields of connective tissue.

Once the histologic diagnosis of juxtacortical osteogenic sarcoma is made, the treatment of choice is amputation of the limb with an adequate margin of uninvolved tissue. Though the temptation to procrastinate in favor of local excision is ever recurring in the clinician's mind, experience by Jaffe and others indicates that ultimately some of these lesions metastasize. Local recurrence, often in a more malignant form, is to be expected when inadequate local removal of the tumor is attempted.

SUMMARY

The cases of juxtacortical osteogenic sarcoma reported in the American literature have been briefly reviewed and a typical case presented with special emphasis on the radiopathologic correlation. Especially in rare and difficult clinicopathologic entities such as this tumor, the best therapeutic results can be obtained only by the close cooperation between the clinician, radiologist, and pathologist.

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MANDIBULOFACIAL DYSOSTOSIS (TREACHER COLLINS SYNDROME)*

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THE syndrome of mandibulofacial dysostosis (Treacher Collins syndrome) concerns itself with a rather unusual and apparently closely related group of congenital defects of the head and face. It has received little attention in radiologic literature, although many of the cases described in ophthalmologic and maxillofacial papers have been substantially documented by roentgen examinations. The syndrome is of considerable interest to the oral surgeon, ophthalmologist, otolaryngologist and maxillofacial surgeon, to whom roentgen studies furnish invaluable aid in determining the extent of associated defects. It is the purpose of this paper to present a brief historic review, together with a description of the clinical and roentgen findings of this syndrome, and to report a case which we examined in the Department of Radiology, Hospital of the University of Pennsylvania.

HISTORIC REVIEW

In 1889, a British ophthalmologist, G. A. Berry,[1] reported a case of congenital defect of the right lower eyelid which he termed a ? coloboma. Although he mentioned that the patient's twin brother had a harelip, he made no note of other congenital deformities in his patient (a young girl). However, an inspection of the sketch which he had made of this patient's head indicates evidence of a slightly receding chin. He did state that the mother of this girl had the same defect as her daughter in the lower lid of both of her eyes, *absolutely symmetrically*, and in addition she had had a harelip which had been operated on. In 1900, E. Treacher Collins,[3] who also was a British ophthalmologist, presented 2 cases of congenital symmetric notching of the outer third part of each lower eyelid, associated with deficient formation of the malar bones. Three similar cases were reported in 1943 in the *British Journal of Ophthalmology* by Mann and Kilner,[9] and Johnstone.[7] In addition to the eyelid defects and malar bone deficiencies, these authors noted micrognathia in all 3 patients. Kilner's patient also had a cleft soft palate and showed bilateral ear deformities.

In 1944, Franceschetti[5] and Zwahlen reporting from the Ophthalmologic Clinic of the University of Geneva added 1 case presenting the complete syndrome, and a second case of the atypical form. They also collected 7 other typical cases recorded in the literature. They felt that all these congenital developmental defects were associated, and suggested the term "dysostosis mandibulo-facialis" for this very peculiar hereditary syndrome.

In 1945, Leopold, Mahoney and Price[8] presented a paper in the American ophthalmologic literature regarding a mother, daughter and grandfather with symmetric lower lid deformities and abnormalities of the zygomatic processes of the temporal bones.

In 1949, Straith and Lewis[10] from the Straith Clinic for Plastic Surgery, Detroit, described in the American literature a family of 5, all of whom exhibited the classic deformities of the syndrome; in addition all had an abnormally long second metatarsal bone. These authors stated the syndrome was referred to in England as the "Treacher Collins syndrome," and they gave the title to their report as Associated Congenital Defects of Ears, Eyelids and Malar Bones (Treacher Collins syndrome).

In 1949, Franceschetti and Klein[6] published a comprehensive review entitled "The Mandibulo-Facial Dysostosis: a New

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Hereditary Syndrome," in which they summarized the findings of most of the previously reported cases in the various countries, and to which they added 3 new cases.

One of the first roentgenologic reports was that of Campbell[2] (from the Scientific Exhibition of the Annual Congress of the British Institute of Radiology, December, 1953) published in 1954, in which he described 2 patients with the Treacher Collins syndrome and summarized the important classic clinical and roentgen findings.

REPORT OF A NEW CASE

A.T., a fifty-two year old white female, was admitted to the Gynecologic Service of the Hospital of the University of Pennsylvania in September, 1955, for the investigation of vulvovaginal edema. Although this was her fourth admission since 1945, no special attention had been directed to her congenital developmental abnormalities during three previous hospitalizations. Upon her last admission, however, we were impressed by the patient's most unusual facial appearance, as well as by other allied clinical and roentgen findings, all of which we felt represented the classic manifestations of the syndrome of mandibulofacial dysostosis (Treacher Collins syndrome).

FIG. 1. Patient A.T. Lateral roentgenogram (underexposed). There is underdevelopment of the maxillary sinuses, with hypoplastic zygomatic bones and recessive mandible. With such a study one can readily understand the facial characteristics exhibited by the patient.

FIG. 2. Same patient. Lateral roentgenogram (underexposed). The relationship of the recessive mandible to the soft tissues of the neck is demonstrated.

Examination revealed that the palpebral fissures were anti-mongoloid, sloping downward and outward. A small notched defect was seen at the outer margin of each lower eyelid, near the external canthus. The eyelashes and puncta were deficient in the lower eyelids. Her chin was moderately recessive. There was a marked deformity of the pinna of the right ear, associated with atresia of the external auditory canal on the same side. The left ear was somewhat less deformed, and the external auditory canal was patent, though stenotic. There was complete loss of hearing on the right side, with some impairment on the left, although bone conduction was normal bilaterally. The temporal hairline extended in a tongue-like projection over the cheeks in front of the ears. The malar eminence was deficient bilaterally. Her mouth was small, and the palate presented a high arch, but no cleft was apparent. No visual defects were present. The patient was of normal intelligence. No other malformations were noted clinically.

The roentgenographic examination (Fig. 1, 2 and 3) showed marked hypogenesis of the

Fig. 3. Same patient. Posteroanterior roentgenogram. Note the marked disparity in the size of the normal calvarium as compared to the underdeveloped mandible. The hypoplastic maxillary sinuses are again visualized.

mandible. The malar bones and maxillary sinuses (Fig. 4) were severely underdeveloped. The mastoids (Fig. 4) were infantile in appearance. The auditory ossicles were thought to be absent, but this observation could not be substantiated. The orbits were normal, as was the calvarium. The normal calvarium (Fig. 3) served to emphasize the underdeveloped mandible by comparison. No other skeletal deformities were detected.

The patient does not recall the existence of any of the above defects in her parents or siblings. She remarked that she had been aware of her peculiar "bird-face" appearance ever since she was a youngster, and that she was deaf in her right ear from birth.

DISCUSSION

Clinical Findings. In their comprehensive review, Franceschetti and Klein[6] identified various signs which they felt were common to all classic cases of mandibulofacial dysostosis (the complete form). Other cases presented one or more of these signs, and were considered incomplete, abortive, unilateral, or atypical. They listed the following important clinical signs, most of which we were able to distinguish in our patient:

1. Anti-mongoloid palpebral fissures with a coloboma of the outer portion of each lower lid, and deficiency of the

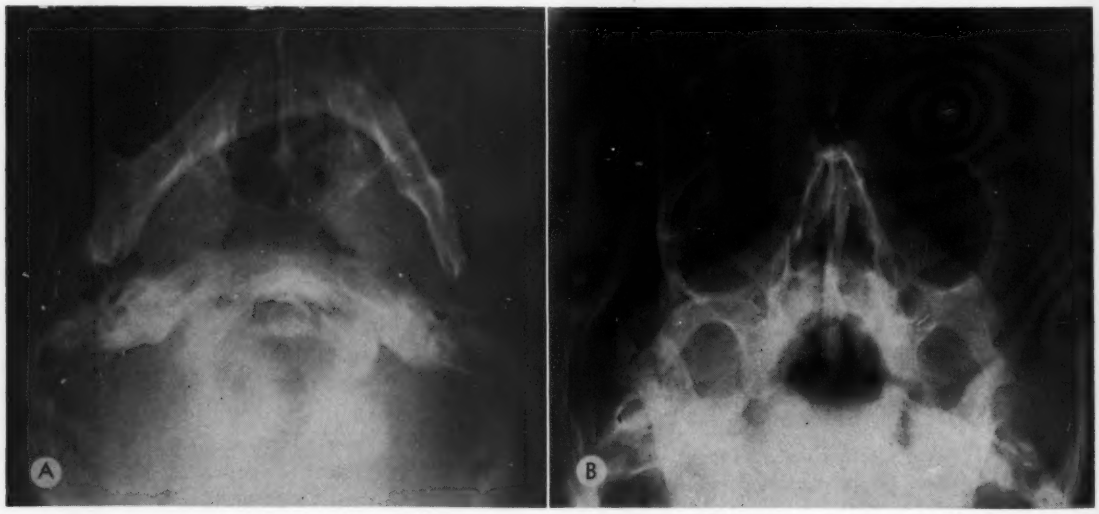

Fig. 4. Same patient. Roentgenograms in the Hirtz (*A*) and Waters (*B*) projections. The small facial bones are readily visualized with hypoplastic maxillary sinuses and underdeveloped zygomatic bones. A similar degree of hypoplasia is noted in the temporal bones.

eyelashes (and more rarely in the upper lids).
2. Hypoplasia of the facial bones, especially of the malar bones and the mandible.
3. Malformation of the external ear, and occasionally of the middle and internal ears.
4. Macrostomia, high palate (sometimes cleft) and abnormal position and malocclusion of the teeth.
5. Blind fistulas between the angles of the ears and the angles of the mouth.
6. Atypical hair growth in the form of a tongue-shaped process of the hair-line extending towards the cheeks.
7. Other additional anomalies such as facial clefts and skeletal deformities.

Classification. Using the above criteria, Franceschetti and Klein[6] classified all cases they found recorded in the literature in the following manner:

1. Complete, or typical forms (standard types); manifesting all the above signs.
2. Incomplete forms; having the same findings, but less strikingly, with almost normal appearance of the ears, and impaired hearing.
3. Abortive forms; showing only one defect (anomalies of the lid only).
4. Unilateral forms; exhibiting underdevelopment of facial bones on the right side and other skeletal deformities.
5. Atypical forms with irregular findings.

As stated previously, our case presented most of the classic features, and although we were unable to detect blind fistulas or other skeletal deformities, we feel that it is truly representative of the mandibulofacial dysostosis syndrome.

Roentgen Findings. The recorded cases demonstrated that the roentgen examination will often reveal the bodies of both malar bones to be grossly and symmetrically underdeveloped. There may be agenesis of the malar bones, with nonfusion of the zygomatic arches, as well as absence of the palatine bones. Cleft palate may be visible on the roentgenogram. There is usually hypogenesis, and sometimes agenesis, of the mandible. The paranasal sinuses are grossly underdeveloped, and the mastoids appear infantile and sclerotic. The auditory ossicles are often absent, and the cochlea and vestibular apparatus may be deficient. The cranial vault is normal in most instances. There may be roentgen evidence of other far-removed skeletal abnormalities, but in view of the very nature of the embryologic deficiencies to be mentioned below, we feel that the pertinent findings in this syndrome are confined to the head and face.

Natural History. Although our patient showed no evidence of familial occurrence, the literature is well documented with many hereditary and familial instances of this syndrome. Leopold *et al.*[8] showed its presence in a mother, daughter, and grandfather, while Straith and Lewis[10] reported a mother and her 4 children, all of whom had the characteristic classic deformities. Debusmann[4] recorded 9 cases in three generations.

Genetically, the syndrome appears to follow an irregular form of dominant transmission.

Mann and Kilner[9] in discussing a patient which they reported in 1943, gave an excellent review of the embryology of the skull and the face, especially as it pertained to the areas involved in mandibulofacial dysostosis. From their study they concluded that the syndrome resulted from a retardation or failure of differentiation of maxillary mesoderm at and after the 50 mm. stage of the embryo. Mann and Kilner have pointed out that when the embryo develops, the paraxial mesoderm surrounding the central nervous system extends in the form of enveloping capsules around the sense organs (olfactory, optic and otic), while the visceral mesoderm advances secondarily in the form of the maxillary process the first visceral arch (mandibular) to produce the supporting skeletal and outer coverings necessitated by the great increase in width of the brain in the higher verte-

brates. The visceral mesoderm forms, in the first place, the wedge of tissue which extends upwards behind the laterally placed eyes to form the temporal bone and muscle. It is mechanically responsible for the swinging around of the eyes from a lateral to forward position necessary in binocular vision. If the differentiation of this wedge is defective, deafness results from improper development of the temporal bones. Secondly, this wedge is responsible for development of the maxilla and malar bones; and finally, its superficial extensions account for the lower lid, the vestibule of the nose and the whole moulding of the side of the face, including the external ear.[9] The first rudiments of lashes and meibomian glands in the lower eyelid appear at the 50 to 60 millimeter stage of the embryo; these are absent in mandibulofacial dysostosis. Since the teeth of the upper jaw are usually unaffected, and ordinarily are present by the sixth week, this is further evidence of retardation or arrest of differentiation at or after the second month of fetal life. The first visceral arch of the visceral mesoderm also advances secondarily to form the mandible, and again retardation occurs on the same basis.[9]

SUMMARY

1. A case report of mandibulofacial dysostosis (Treacher Collins syndrome) is presented and an historic review of this syndrome is given. It concerns a group of closely related hereditary and familial defects of the head and face.

2. This syndrome has long been familiar to maxillofacial and oral surgeons and to ophthalmologists, but has received little attention in the radiologic literature.

3. The embryology of the skull and face is reviewed which reveals retardation or failure of differentiation of maxillary mesoderm at and after the second month of fetal life.

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ROENTGENOGRAPHIC ABNORMALITIES OF THE SKELETAL SYSTEM IN WILSON'S DISEASE (HEPATOLENTICULAR DEGENERATION)*

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WILSON'S disease is the result of an hereditary defect of metabolism which is associated with an excessive absorption of copper from the intestinal tract and consequent increased copper content of the body.[4] It seems probable that copper deposition in the brain and liver is responsible for the clinical syndrome of hepatolenticular degeneration. The continued, slow accumulation of copper is seen also in other tissues and may be responsible for other manifestations of the disease. During a systematic study of the clinical and biochemical aspects of Wilson's disease, it became apparent that these patients frequently showed roentgenographic changes in their skeletal system. The present study describes the nature and extent of the bone and joint abnormalities in 20 patients with Wilson's disease.

CLINICAL MANIFESTATIONS

The clinical manifestations of Wilson's disease usually appear in young adults, between the ages of twelve and twenty. The predominant symptoms are referable to the nervous system. Tremor, rigidity, dysarthria and dysphagia are a result of disorganization of the lenticular region of the brain. Cirrhosis of the liver is almost always present but is seldom the dominant clinical feature. When the disease appears in adults, it is milder and more slowly progressive than in younger patients.

The Kayser-Fleischer ring, a brown or grayish green ring at the limbus of the cornea, is pathognomonic of Wilson's disease and is seen in over 95 per cent of patients. Excessive quantities of copper have been found in the Kayser-Fleischer corneal ring. Similar deposits of copper in the cerebral cortex, kidney and other tissues[22] may be related to the mental deterioration, the renal changes and the skeletal abnormalities occasionally seen in these patients.

LABORATORY FINDINGS

Recent studies have shown that there is an increased urinary excretion of copper in patients with Wilson's disease.[5] Nevertheless, the serum copper is usually strikingly decreased, although normal values may sometimes occur.[5,9] Most (95 per cent) of the serum copper is bound to the serum globulin, ceruloplasmin. In Wilson's disease the serum ceruloplasmin shows a proportionately greater decrease than the serum copper and more of the serum copper is bound to albumin (direct reacting copper) than in normal patients.[9,19] These characteristic biochemical findings allow a diagnosis of Wilson's disease, even in those patients in whom the classic symptoms are absent.

HEREDITARY ASPECTS

Studies of the genetic aspects of Wilson's disease indicate a recessive mode of inheritance; the affected patients are homozygous for a rare recessive gene. The first cousin consanguinity rate of the parents of 26 patients studied at The Rockefeller Institute is fifty times as high as that reported in a general hospital population.[4] The disease is particularly common in those ethnic groups in which a high incidence of consanguinity normally prevails. Eighty per cent of the 26 patients were either Jewish or Italian.[3]

RENAL ABNORMALITIES

Many patients with Wilson's disease show an increased urinary amino acid ex-

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Fig. 1. Osseous lesions in a young man with Wilson's disease of three years' duration. (J.M., Case 16 Table 1). (*A*) Hand and wrist at age of fifteen years showing bone demineralization and epiphyseal changes of severe osteomalacia (rickets). (*B*) Hand and wrist at age of seventeen years showing progression of osteomalacic change.

cretion.[10] Although the aminoaciduria involves a broad spectrum of amino acids, the excretion of threonine and cystine is strikingly increased. Further studies of kidney function have shown defective tubular reabsorption of phosphate with a consequent increased urinary excretion of phosphate. This is usually associated with a low plasma phosphate level. A systematic study of renal function in patients with Wilson's disease suggested a striking similarity to the renal lesion found in the Fanconi syndrome.[6] Renal clearance studies indicated a progressive deterioration of certain discrete tubular functions. All these changes may be reasonably ascribed to the deleterious effects of copper deposition in the kidney.[6]

BONE AND JOINT ABNORMALITIES

Warnock,[21] in 1952, noted spontaneous fractures and osteoporosis in one of his patients with Wilson's disease. Similar instances had been noted by von Economo,[12] Brückner,[8] Kehrer,[14] Luthy[15] and André.[1] Cooper *et al.*[10] described a patient with Wilson's disease in whom increased aminoaciduria, derangement of calcium and phosphorus metabolism and osteomalacia were demonstrable. These isolated reports suggested a possible relationship between the skeletal abnormality and the underlying disease. Since the increased urinary excretion of phosphate and the low plasma phosphate level in some patients with Wilson's disease might explain this relationship, we undertook a systematic roentgenographic study of the skeletal structures in our patients.

Of 20 patients with Wilson's disease in whom a roentgenographic skeletal survey was accomplished, only 6 had normal osseous structures. The major skeletal abnormalities are summarized in Table 1.

Osteomalacia and Milkman Pseudofractures. Seven of the 20 patients (Table 1)

TABLE I

Case No.	Name Age Sex	Approximate Duration of Overt Disease (Years)	Clinical Severity[1]	Osteomalacia	Milkman Pseudofracture	Bone Fragmentation	Osteoarthritis	Fractures	C_{In}[2] ml./min.	P_{PO_4}[3] mg. per cent	C_{PO_4}[4] ml./min.	C_{PO_4}/C_{In}[5] per cent	P_{Ca}[6] mg. per cent
1	MF, 40, M	3	+++	o	o	o	+	+	91.9	2.4	28.0	30.4	9.0
2	RV, 13, M	4	+++	o	o	o	o	o	107.0	3.3	37.1	34.7	11.1
3	MG, 33, M	5	+	o	o	+	+	o	89.7	2.5	22.4	25.0	10.0
4	PC, 36, F	7	++	o	o	+	+	o	76.2	2.5	18.7	24.5	9.1
5	LA, 53, M	20	+	+	o	+	+	+	48.9	1.5	28.8	59.0	10.7
6	YR, 37, F	23	++	+	o	+	+	+	63.5	2.5	19.5	30.7	10.1
7	CP, 27, M	6	+	o	o	+	+	o	85.7	3.2			
8	FL, 35, M	8	+	o	o	+	+	o	83.2	3.1			9.4
9	BR, 37, F	23	++	+	o	+	+	o	69.0	2.7			10.7
10	RG, 41, M	8	++	+	+	o	+	+		3.2			9.1
11	VR, 16, M	2	++	+	o	o	o	o					
12	CR, 29, F	8	++	o	o	o	+	+					
13	WC, 30, M	14	++	o	o	o	o	o		3.1			
14	TM, 18, M	3	+	+	o	+	o	+		3.1			8.6
15	GV, 31, M	3	+	o	o	o	o	o					
16	JM, 17, M	3	+++	+	+	o	o	+					
17	MA, 44, F	14	+	o	o	+	+	o		1.5			9.7
18	FM, 32, M	5	+	o	o	o	o	o		3.0			10.1
19	JMcD, 20, M	3	+++	o	o	o	o	o		3.1			10.5
20	IS, 45, M	12	+	o	o	o	o	o		3.0			

[1] +—Mildly incapacitated.
++—Severely incapacitated.
+++—Bedridden.
[2] Normal M 124, F 108.
[3] Normal 3–5.
[4] Normal 10–12.
[5] Normal 10.0.
[6] Normal 9–10.5.

showed evidence of osteomalacia which varied from severe bone demineralization to patchy areas of bone rarefaction. One seventeen year old patient (Case 16, J.M.) showed advanced osteomalacia (Fig. 1) with widening of the epiphyseal line and severe demineralization indicative of "rickets." Skeletal maturation was retarded. These changes resemble those described by Baxter and Van Wyk[2] in young dogs made severely copper deficient by dietary means.

Two patients showed Milkman pseudofractures characterized by radiolucent bands in bone with little or no callus formation (Fig. 5). These were limited in number and asymmetric. However, the roentgenographic appearance was similar to the zones of reconstruction (Umbauzonen) described by Looser in 1920 and Milkman[17] in 1934. These osteoid zones are seen in many patients with acute osteomalacia,[16] and are frequently observed in patients with the adult Fanconi syndrome.[20]

Two patients showed areas of bone resorption localized to the medial ends of the clavicles (Fig. 7). These areas probably represent localized osteomalacic change since they were not associated with clinical evidence of trauma or infection.

Bone Fragmentation. Nine patients showed an unusual bone fragmentation at joint margins (Fig. 2–4 and 6). This was occasionally, but not always, associated with advanced osteoarthritic change. The marginal bone fragmentation was seen most commonly in the wrists and hands but was also observed in elbows, shoulders, hips and knees.

Patients with Wilson's disease, because of their spasticity and tremors, are more liable to minor injuries than normal persons. Thus, chronic trauma could be responsible for the cartilage damage and bone fragmentation. However, the possibility of intrinsic cartilage disease with subchondral bone rarefaction and fracture appears more

Fig. 2. Osseous lesions in a thirty-three year old man with Wilson's disease of five years' duration (M.G., Case 3, Table 1). (*A*) Hands and wrists showing marginal bone fragmentation (arrows). Note the osteoarthritic changes in the wrist and thumbs and the mild subchondral bone irregularity in many of the metacarpophalangeal joints. (*B*) Examination of the right elbow shows bone fragmentation (white arrows), severe osteoarthritis and soft tissue calcification (black arrow). (*C*) Lumbar spine shows severe osteoarthritic change, diskogenic disease and marginal fragmentation of osteophytes.

likely since similar bone fragmentation has not been seen in patients with other tremulous neurologic disease such as Parkinson's disease or multiple sclerosis. Additional evidence in favor of intrinsic cartilage abnormality is the unusual subchondral bone irregularity in the hands of 2 sisters with Wilson's disease. This striking marginal bone destruction at joint surfaces is shown clearly in Figures 3 and 4.

Osteoarthritis. Cartilage injury was noted in 11 of 20 patients in whom severe osteoarthritis was present (Fig. 2–4 and 6). Eight of the 11 were forty years of age or younger and exhibited more advanced osteoarthritic changes than are usually seen in this age group. Bohatirchuk[7] indicates that 35 per cent of men and 30 per cent of women below the age of forty years might be expected to show changes of osteoarthritis, but the changes are quantitatively less pronounced than in the older age group. The large exostoses, the marked thinning of joint spaces and the prominent marginal sclerosis due to osteoarthritis in the patients with Wilson's disease are too advanced to be readily explained by aging, trauma or tremors.

Other Osseous Abnormality. The tendency for patients with Wilson's disease to sustain fractures has been noted in many case reports.[1,8,12,14,15,21] Seven of our 20 patients

Fig. 3. Osseous lesions in a thirty-seven year old woman with Wilson's disease of twenty-three years' duration (Y.R., Case 6, Table 1). Hands show pronounced subchondral bone irregularity and destruction involving all metacarpophalangeal joints. Note the bone fragmentation and severe osteoarthritis in the wrists.

exhibited evidences of fractures during the course of their disease. The increased likelihood of trauma in Wilson's disease may be associated with an increased bone fragility due in instances to a disturbed phosphorus metabolism.

Incidental findings included osteomata or areas of nonreticulated bone (bone islands) which were seen in 7 patients. Hyperostosis frontalis interna of moderate or marked degree was noted in 4 women patients.

DISCUSSION

It is of interest that 14 of 20 patients with Wilson's disease showed evidence of significant skeletal abnormality. This was manifested by roentgenographic evidences of osteomalacia, cartilage injury or bone fragmentation.

An attempt has been made to relate the osseous abnormalities to the renal changes of Wilson's disease. The first six patients in Table 1 were studied in detail by Bearn, Yü and Gutman.[6] These patients showed an impaired urinary inulin clearance (C_{In}) and an increased phosphate clearance (C_{PO_4}). The ratio C_{PO_4}/C_{In} indicates the relative severity of the defect in phosphate reabsorption and is reflected by the low normal values of plasma phosphorus. The plasma calcium values were usually within normal limits.

The osteomalacia and Milkman pseudofractures in some patients with Wilson's disease may be related, in part, to the described abnormality in phosphorus metabolism. A similar association is seen in patients with the Fanconi syndrome.[20] This syndrome is characterized by osteomalacia with multiple fractures and pseudofractures, hypophosphatemia, elevated alkaline phosphatase, massive generalized aminoaciduria with normal plasma aminoacid level and renal glycosuria. Many of these

Fig. 4. Osseous lesions in a thirty-seven year old woman with Wilson's disease of twenty-three years' duration (B.R., Case 9, Table 1). This patient is a twin sister of Y.R., Case 6 (See Figure 3). Hand and wrist show severe osteoarthritis and bone fragmentation, most marked in wrist. Note subchondral bone irregularity of metacarpophalangeal joints.

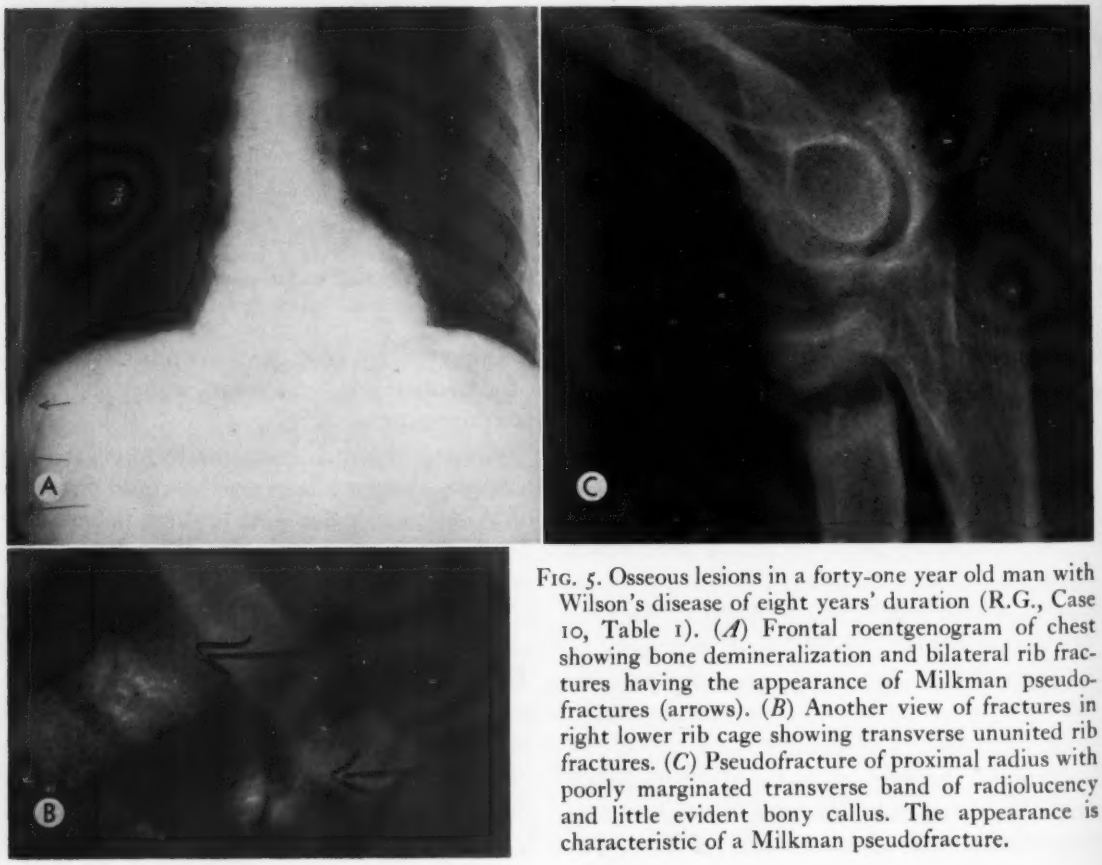

Fig. 5. Osseous lesions in a forty-one year old man with Wilson's disease of eight years' duration (R.G., Case 10, Table 1). (*A*) Frontal roentgenogram of chest showing bone demineralization and bilateral rib fractures having the appearance of Milkman pseudofractures (arrows). (*B*) Another view of fractures in right lower rib cage showing transverse ununited rib fractures. (*C*) Pseudofracture of proximal radius with poorly marginated transverse band of radiolucency and little evident bony callus. The appearance is characteristic of a Milkman pseudofracture.

Fig. 6. Osseous changes in a fifty-three year old man with Wilson's disease of twenty years' duration (L.A., Case 5, Table 1). Bone fragmentation and osteoarthritic changes are seen in the wrists. Hands show mild bone demineralization. Note small areas of nonreticulated bone (bone islands) in many of the phalanges.

features are evident in patients with Wilson's disease. Thus, as suggested by Wallis and Engle[20] and Dent and Harris,[11] Wilson's disease may be another hereditary source of the Fanconi syndrome.

It is unlikely that all the skeletal abnormalities found in patients with Wilson's disease can be related to the disturbed renal function. Moreover, the bone fragmentation, osteoarthritis and cartilage degeneration are less easily explained than the osteomalacia or pseudofractures. Thus, the possibility that the skeletal abnormality may be directly due to the abnormal copper metabolism must be considered and warrants further study.

Skeletal abnormality due to intoxication with other heavy metals is well known. The roentgenographic manifestations of lead, bismuth, radium, strontium and fluorine poisoning are due to their deposition in bone. Nicaud, Lafitte and Gros[18] describe symmetric pseudofractures in the bones of cadmium workers who had inhaled vapors of cadmium oxide for many years.

The exact relation of copper metabolism to bone disease is not known. Gubler[13] points out that studies with intravenous Cu 64 show less copper concentration in bone and muscle than in the liver and nervous system. However, because of their total mass, the muscles and bones contain more than 50 per cent of the total body copper. Baxter and Van Wyk[2] have shown that copper is essential, either directly or indirectly, for normal bone formation since a deficiency of copper in dogs results in thin, malformed bones which show a lack of normal

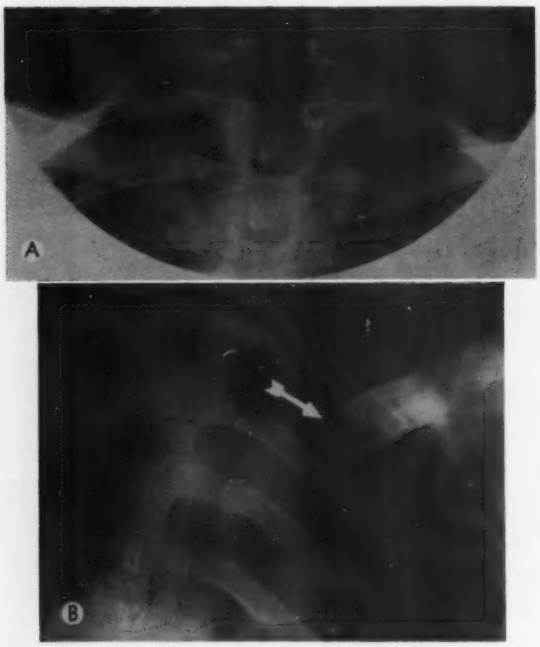

Fig. 7. Osseous change in a sixteen year old boy with Wilson's disease of two years' duration (V.R., Case 11, Table 1). This patient is a brother of C.R., Case 12. (*A*) The medial ends of both clavicles, especially the left, show a poorly marginated area of bone demineralization or destruction. (*B*) Oblique projection allows a clear view of the ragged margin at the medial end of the left clavicle (arrow).

mineralization and trabeculation. It is not immediately apparent how these observations in animals are related to the present findings in patients with Wilson's disease in whom an increased total body copper is ordinarily found. Comparative studies on the copper content of cartilage in normal subjects and in patients with Wilson's disease are now in progress.

SUMMARY

1. Fourteen of 20 patients with Wilson's disease were shown to have a variety of osseous lesions. Seven patients showed evidence of osteomalacia; 2 of these had Milkman pseudofractures. Nine patients exhibited an unusual marginal bone fragmentation and 11 showed roentgenologic evidence of advanced osteoarthritis despite their relative youth.

2. The similarity between certain aspects of Wilson's disease and the Fanconi syndrome, in which bone abnormalities have been well documented, is discussed. The limitation of the hypothesis that the bone lesions in Wilson's disease are related to the disturbance of phosphate metabolism is emphasized. It is possible that the abnormal copper metabolism is itself related to the skeletal abnormalities.

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We would like to express our thanks to Dr. C. E. Dent who drew our attention to the report of bone lesions in cadmium poisoning[18] and to Dr. J. H. Baxter[2] who furnished the original roentgenographic examinations of his copper deficient dogs for our study.

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PSORIATIC ARTHRITIS

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PSORIATIC arthritis as a clinical entity was first presented by Alibert in 1822. Since that time several papers have been published both in this country and abroad. It is interesting to note, however, that the largest number of radiological contributions have been in the foreign literature and only two in American radiology journals. The remainder of the American works on the subject is to be found primarily in medical or medical specialty journals. We feel that further emphasis on the roentgenologic aspects of this condition would be desirable.

We have recently seen a case of psoriatic arthritis in this department which differs from those previously reported, both in age of the patient and type of lesions. Our case also presents one variety of lesion which to our knowledge has never been reported, but which may well be of clinico-pathologic significance. A second case is also presented which is more in keeping with those previously described, being in an older individual, with less marked joint changes.

Psoriatic arthritis is very easily confused with other types of arthritis, particularly the rheumatoid type, or with those associated with trophic disturbances. Only by constantly bearing the entity in mind and by closely correlating the clinical and roentgenographic findings can the diagnosis be made.

As one reads the available literature on the subject, it appears that this is a disease of the older age group, although Epstein,[2] on clinical evaluation, cites a peak incidence between twenty and thirty years. Even in his series, however, he lists several cases over fifty years of age. Certainly the majority of articles in the radiological literature deals with elderly patients. In many of the previously reported cases the diagnosis is somewhat confused because, being in the older age group, there is associated unrelated pathology which clouds the roentgenographic as well as the clinical picture. Also, because of the variation in definitions of the disease, cases described as psoriatic arthritis by one author are not considered as such by another. Roxburgh[6] in his dermatology text has described psoriatic arthritis as "a severe form of the disease in which the attacks are accompanied by a form of periarthritis resembling rheumatoid arthritis, which waxes and wanes in company with the eruptions on the skin." This obviously is a clinical definition, but because it includes the prominent clinical features, without being too specific, I believe that it is the best definition. Other writers have been more exacting and have set rigid criteria for the type and area of involvement, so that their definitions would rule out a good percentage of the reported cases.

The majority of authors agrees that this disease may be polyarticular in nature and there is unanimous accord that the cutaneous form of the disease is present for many years prior to the onset of the arthritic phase. In many cases the onset of the arthritis is accompanied by an extension of the cutaneous lesions which often develop into the pustular form of the disease, at the same time becoming more refractory to therapy.

The roentgenographic findings in psoriatic arthritis are essentially those of a trophic arthritis. Brailsford[1] gives a fairly complete account of these changes, which may be briefly summarized as follows: initial osteoporosis followed by increased density of the involved bones; diminished joint space and erosion of the joint surfaces with atrophy of the shafts; and finally, in advanced cases, essentially complete destruction of the extremities of the bones so that they are atrophic and tapered.

The therapy of cutaneous psoriasis has been discussed at great length, and no single medication has been found effective in curing the disease. In psoriatic arthritis

several methods of treatment have been suggested, including local roentgen therapy, roentgen therapy to the sella turcica (because associated sellar enlargement has been described), local heat and others. None of these has proved very effective, as is obvious from the large number of methods suggested. Fawcitt[3] has recommended the use of cortisone, explaining its possible beneficial effect on the basis of the alarm reaction theory propounded by Selye. One of our patients is being treated with meticorten, and for a time the results were very gratifying, but symptoms have recurred even though the patient has continued taking the drug.

REPORT OF CASES

Case 1. R.S., a nineteen year old white male, had a history of cutaneous psoriatic lesions of five years' duration. The lesions cleared during the summer months and recurred in the winter. Two years ago, with the exacerbation of the skin lesions, he noted pain and swelling of the feet, and despite therapy by an orthopedic surgeon, the symptoms persisted until spring, when they subsided spontaneously. Again in October, however, with the onset of cold weather, the symptoms returned, accompanied by right ankle pain and swelling. At this time roentgenograms of the foot were taken, and the patient was put on colchicine as a therapeutic test for gout. When he received no relief from this therapy he was hospitalized for further study. On admission he complained of pain and swelling of the right third finger in addition to the symptoms already mentioned.

Physical examination revealed typical psoriatic patches on both knees, elbows, dorsum of hands, between the fingers and on the scalp. No nail involvement was noted. The proximal phalanx of the right third finger was swollen and the interphalangeal joint stiff. The right ankle showed some limitation of motion and was swollen. Rather marked atrophy of the muscles of the feet was noted and there was a third degree pes planus. The right fifth metatarsophalangeal joint was somewhat tender and motion was limited.

All routine laboratory examinations were normal. The corrected sedimentation rate was 19 mm. in one hour. Fasting blood sugar and uric acid levels were well within normal limits.

Fig. 1. Case 1. Right fifth meta tarsophalangeal joint.

An electrocardiogram showed a sinus arrhythmia and slight right axis deviation. No explanation for this was found on either physical examination or cardiac fluoroscopy. Throughout his hospitalization the patient felt well, and was treated with aspirin and meticorten.

Roentgenographic examination of the right foot showed destructive changes of the metatarsal head and base of the phalanx in the region of the fifth metatarsophalangeal joint (Fig. 1). The joint space itself was nonexistent, and the distal end of the metatarsal appeared to be taking on the tapered appearance described by Brailsford. The destruction in the proximal phalanx was somewhat less extensive.

Examination of the left foot revealed early changes in the fourth metatarsophalangeal joint (Fig. 2), consisting of partial obliteration of the joint space with mild osteoporosis of the adjacent bone. Early destructive changes of the proximal end of the proximal phalanx were also noted. No roentgenographic changes were seen in the region of the right ankle joint.

Another finding noted in this patient was rather marked periosteal reaction of the proximal phalanx of the right third finger

Fig. 2. Case 1. Left fourth metatarsophalangeal joint.

(Fig. 3). On questioning the patient a history of slight trauma was elicited, this having occurred about six months prior to admission. The subperiosteal ossification has persisted, and there is some associated narrowing of the proximal interphalangeal joint space.

Case 11. J.G., a fifty-six year old white male, had a history of cutaneous psoriasis beginning at twenty years of age with slow progression to the present time. The patient first recalled scaling lesions of the scalp and chest occurring at about twenty years of age. Ten years ago he developed similar changes on the abdomen, in the region of the umbilicus, and about three years ago, lesions of both elbows. The only change noted in these lesions has been a slight increase in their extent in the winter months, with regression during the summer. For many years the patient had noted painless swelling of the fingers during the day without other associated symptoms. In 1945 he developed scaling lesions and irregularity of the right little fingernail, followed in about one year by gradually increasing pain and swelling of the interphalangeal joints of that finger. This has been repeated in other fingers of both hands although the symptoms have been milder. Approximately ten years ago the patient noted pain in the interphalangeal joint of the right great toe. Soon after the symptoms began he noted progressive scaling and defromity of the nail of that toe. Other toes have become involved, particularly the right second toe, and though symptoms have been present in the remainder of the toes of the right foot and most of the toes of the left foot, no other nail involvement has occurred.

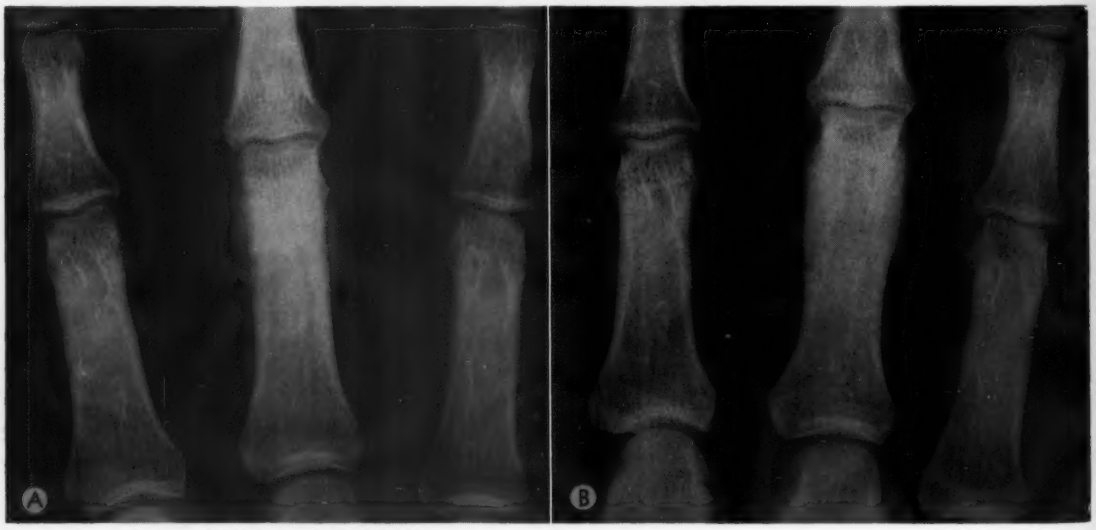

Fig. 3. Case 1. Proximal phalanx of right third finger showing abnormal periosteal reaction. (*A*) May 10, 1955. (*B*) December 28, 1955.

Previous roentgen examinations had not been performed. Despite the long history of joint symptoms, films at this time did not show the striking changes noted in the previous case (Fig. 4 and 5). There was narrowing of all the interphalangeal joints of the feet with the exception of that of the left great toe. No definite evidence of sclerosis was noted; however, moderate destructive changes were seen adjacent to the interphalangeal joints of the right great toe and the distal interphalangeal joint of the right second toe. There was no tapering of any of the bones of the foot, although there was suggestive early tapering of the proximal phalanx of the right great toe. Some narrowing of the first metacarpophalangeal joint spaces was noted but this was of a rather nonspecific nature and could not be closely correlated with the psoriasis.

Roentgenographic examination of the right hand (Fig. 6) revealed less dramatic changes than in the feet, the only definite finding being narrowing of the distal interphalangeal joint space of the fifth finger and minimal loss of detail of the adjacent bones. This loss of detail appeared to be secondary to bone absorption.

Fig. 5. Case II. Right foot showing destructive changes in first and second toes.

Fig. 4. Case II. Left foot showing narrowing of interphalangeal joint spaces.

Fig. 6. Case II. Right hand showing early changes in interphalangeal joints of little finger.

Roentgenograms of the left hand were essentially negative.

This patient has been treated with numerous topical medications for many years without significant change either in symptoms or extent of the cutaneous lesions. Several parenteral medications have also been used, including cortisone, with no improvement in either the skin lesions or joint pain. The only medication which has given any satisfactory degree of relief has been colchicine, though the reason for this relief is not understood. Uric acid determinations have been within normal limits and no other findings consistent with gout have been discovered.

DISCUSSION

Before arriving at a final diagnosis of psoriatic arthritis in the first case there were several findings which had to be correlated with the over-all picture. The condition is generally considered to be somewhat rare at this age, despite Epstein's finding of a peak incidence from twenty to thirty years of age. This patient was nineteen years of age and had a history of cutaneous psoriasis of at least three years' duration prior to the onset of the joint manifestations. Since psoriasis is a disease involving all age groups, it is not unreasonable to assume that some of the younger patients might also have joint involvement. When this joint pathology follows so closely the course of the cutaneous disease, increasing in severity as the skin lesions increase and becoming asymptomatic when the lesions regress, it becomes quite difficult to explain the changes on any other basis. This is especially true when there is no definite evidence suggestive of other pathology. The bony changes illustrated are quite typical of those described by Brailsford, and although they are not specific for this disease, correlation with the clinical picture certainly produced strong evidence in favor of the diagnosis.

The additional finding presented was the rather marked persistent periosteal reaction to minor trauma, with involvement of the joint space of that digit. Again, this finding was not specific for any disease process but might well have been a part of the generalized metabolic disturbance which was present. Although not previously described in this condition, one wonders if one were dealing with another manifestation of the same disease, or with a heretofore unrecognized indication of its systemic nature.

In the second case, we were dealing with a more typical case of bone involvement in an older individual with a very long history of cutaneous psoriasis with less marked and less specific bone changes. Since the 2 cases differed both in clinical course and in physical findings, the question arises whether they might not have been two different forms of the same disease.

One might recall the variation in the course of many diseases in young and old individuals. Diseases such as leukemia, tuberculosis, carcinoma of the cervix or breast, etc., often are rather acute in children or young adults whereas they tend to be more chronic in older individuals. One may postulate a similar division here; namely, a more acute fulminating juvenile form and a slowly progressive chronic adult form of the disease.

Finally, therapy of psoriasis at the present time leaves much to be desired. Many preparations have been tried, but none has been found very effective. One patient was treated with meticorten, and for a time both cutaneous lesions and joint manifestations improved considerably. However, after several weeks, in spite of a continued maintenance dose of the drug, the condition started to progress again, with increase in the size of the skin lesions, and recurrence of the joint symptoms, though to a lesser degree than previously noted. No changes in the roentgenographic appearance of the joints occurred with the clinical changes. Here again, as earlier in the course, the joint symptoms closely paralleled the changes in the cutaneous phase, further supporting the diagnosis of psoriatic arthritis.

Cortisone, on the other hand, was of no

benefit in the second patient. As noted above, colchicine therapy has given the best relief in this patient, though no explanation for this response can be offered. It is fairly apparent however, that much is still to be learned regarding the therapy of this disease, and that the only method available today is that of trial and error.

SUMMARY

A brief discussion of the paucity of American literature concerning psoriatic arthritis is presented.

The criteria for diagnosis of this disease have been demonstrated in 2 patients; namely, cutaneous psoriasis of long duration, parallel exacerbations and remissions of the joint symptoms and the cutaneous lesions and typical atrophic joint changes. The additional finding of marked periosteal reaction following minimal trauma is introduced as a possible related finding in the systemic form of psoriatic arthritis. A second problem posed is the possibility of the existence of two distinct phases of this disease: an acute juvenile and a chronic adult phase. Further study concerning this subgrouping is now in progress.

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BONE LESIONS ASSOCIATED WITH VILLONODULAR SYNOVITIS*

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VILLONODULAR synovitis is a not uncommon disease of synovia, tendon sheaths and bursae which was first described by Simon[9] in 1865 as xanthoma of synovia. From that time until 1941, when the term villonodular synovitis was coined by Jaffe, Lichtenstein and Sutro,[4] this disease was known by a variety of names, partly derived from its gross appearance and partly from the predominant cell type found on microscopic examination.

PATHOLOGY

Villonodular synovitis is seen in two forms. There is a circumscribed form in which single or multiple polypoid masses are attached to the synovium and a diffuse form in which there is hyperplasia of all synovial villi and in which the entire synovial surface has a mossy or bearded appearance. In those of our cases in which bone lesions were present, the disease was of the second or diffuse form.

Microscopically these lesions of synovium, tendon sheath and bursa present a similar picture. They consist of combinations of closely compacted polyhedral stromal cells, giant cells and histiocytes containing cholesterol and hemosiderin, interspersed with areas of collagen and hyaline degeneration. If large numbers of hemosiderin containing cells are present, the mass is grossly dark in color, and the term pigmented villonodular synovitis is used. A more detailed description of the microscopic findings is given by Jaffe *et al.*,[4] who concluded from their studies that this is an inflammation rather than a neoplasm as is suggested by the previously used names such as: xanthoma, xanthogranuloma, giant cell tumor of synovia or tendon, giant cell fibrohemangioma, myeloplaxoma, fibrohemosideritic sarcoma, etc.

ETIOLOGY

The etiology of villonodular synovitis is uncertain. Because a history of trauma or repeated trauma is frequently obtained, it has been suspected that chronic or repeated hemorrhage within a joint may be responsible. The term chronic hemorrhagic synovitis has been used to describe the diffuse form. The theory of "chronic hemorrhage" receives support from an experiment by Young and Hudacek[11] who injected the joints of animals with autogenous unclotted fresh blood and reported the production of lesions grossly and microscopically resembling pigmented villonodular synovitis. A similar experiment reported by Jaffe *et al.*[4] failed to produce this lesion.

BONE LESIONS

There are only a few reports of bone lesions associated with intra-articular villonodular synovitis in the literature.[1,6,12] The bone lesions associated with villonodular synovitis of tendon sheaths are quite well known. The subject of intra-articular villonodular synovitis was reviewed by De Santo and Wilson[2] in 1939, who found 32 published cases and added 9 of their own, by Jaffe and co-workers[4] in 1941, who added 20 cases, and by Shafer and Larmon[7] in 1951, who described 7 cases. None of these reports stressed the occurrence of distinct bone lesions. It is our opinion that the bone lesions are initially due to pressure from the soft tissue mass. This pressure causes erosion of the articular cartilage and of the cortical bone; the cystic areas within the bone are produced by expansion of the mass in the softer cancellous bone. Although the most common site of villonodular synovitis is at the knee joint, roentgenographically demonstrable erosions

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Fig. 1. Case 1. Villonodular synovitis of the tendon sheath of the second toe with bone erosion and cyst formation in the middle phalanx. Discomfort due to the large soft tissue swelling was the main complaint of the patient. (Reproduced by permission.[6])

or cysts have not been reported at this site apparently because the large easily distended synovial space allows the mass to expand without causing undue pressure on articulating surfaces or cortical bone. One case[7] is reported in which minor cartilage and bone erosions of the patella and lateral femoral condyle were found at operation for a recurrent villonodular synovitis, seven years after a previous partial synovectomy. The bone lesions were apparently too small to be seen on roentgen examination.

ROENTGENOGRAPHIC FINDINGS

The roentgenographic appearance of villonodular synovitis was discussed by Lewis[5] in 1947. Since he was describing the disease at the knee joint he stressed the absence of bone lesions; however, the other diagnostic criteria he proposed are still pertinent. They are: (1) monarticular involvement; (2) excessive synovial swelling, particularly diagnostic if it has a nodular or lobulated appearance; (3) absence of calcification within the synovial swelling; (4) normal mineralization of the bones of the involved joint; (5) preservation of the cartilage space of the joint.

Lewis stated that the synovial masses must be confined to the joint space; however, extension of the synovial granulomatous tissue through the joint capsule and into adjacent muscles and tendons has

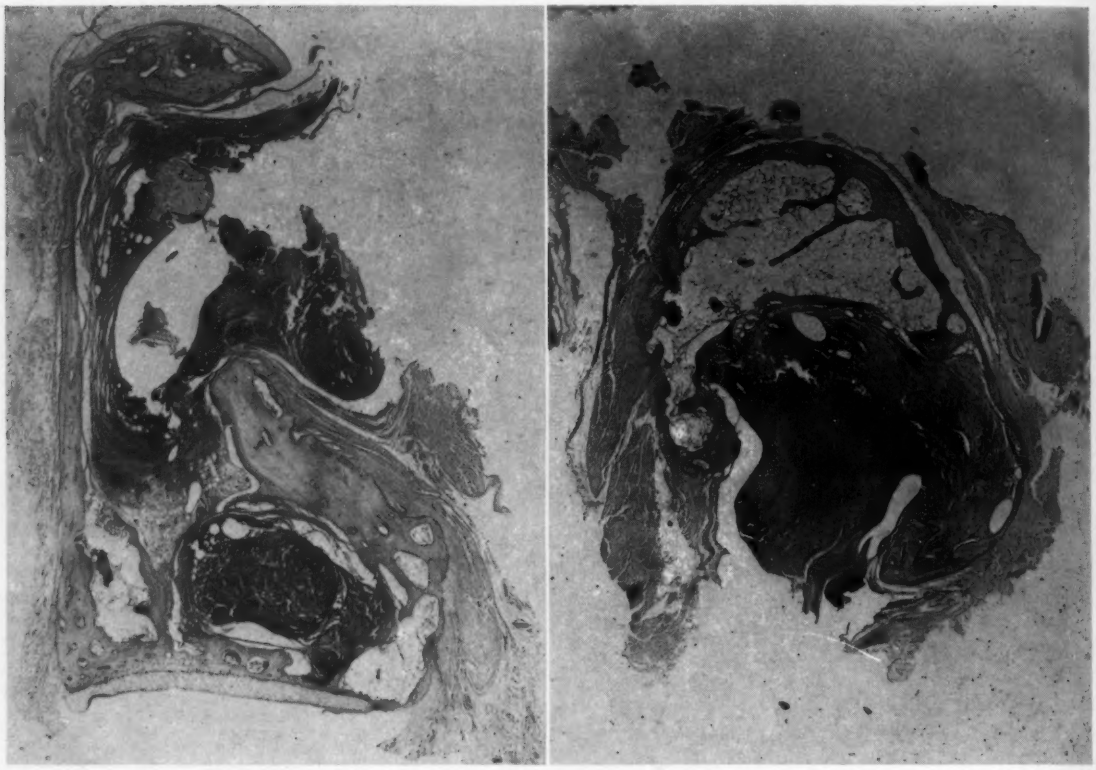

Fig. 2. Case 1. Longitudinal and cross section through the amputated middle phalanx. Erosion of cortical bone and expansion of the granulomatous tissue in cancellous bone are demonstrated.

been reported[1] and was seen in 2 of the cases reported below. Bone lesions appear at the involved joint as sharply circumscribed erosions and single or multiple cysts having thin sclerotic margins. Although there are numerous other diseases in which bone erosions are seen in conjunction with soft tissue masses, erosions and single or multiple cysts in bone adjacent to an articular surface with a noncalcified lobulated intra-articular mass, normal bone mineralization and preservation of the joint space appear to be characteristic of villonodular synovitis.

TYPICAL HISTORY AND CLINICAL FINDINGS

The typical history of villonodular synovitis is increasing swelling of a single joint over a period of months or years in a young or middle aged individual. There is no pain in the early stages and only moderate pain or discomfort is present in the later stages. There may be a history of initial injury or repeated injury.

Physical examination shows a swollen joint with little tenderness and little if any limitation of motion. Aspiration of the joint produces hemorrhagic or xanthochromic fluid with rapid re-accumulation of fluid after aspiration. The fluid has a high cholesterol content but there is no hypercholesteremia or other findings of systemic disease. The patient has no fever or leukocytosis and the sedimentation rate is normal. Tendon sheath lesions, most commonly seen in fingers and toes, show a progressive swelling with little pain or limitation of motion.

DIFFERENTIAL DIAGNOSIS

The differential diagnosis of villonodular synovitis includes tuberculous arthritis and caries sicca, synovioma, osteoarthritis, rheumatoid arthritis and gout.

Fig. 3. Case 11. Villonodular synovitis of tendon sheath of the middle finger, showing minimal bone involvement associated with a mildly painful soft tissue mass.

Ruling out tuberculosis may be quite difficult, particularly tuberculosis sicca which may produce multiple cysts in subchondral bone with relatively good preservation of the cartilage space. This disease is usually not associated with roentgenographically demonstrable synovitis or synovial masses. Ordinary tuberculous arthritis with its marked synovitis also shows marked osteoporosis and often there are clinical findings of fever, elevated sedimentation rate and leukocytosis which are absent in villonodular synovitis. The tuberculin test is of importance only when it is negative.

Synovial sarcoma frequently has lime deposits within its soft tissue masses, a finding which has not been reported in villonodular synovitis. Bone lesions, when present, are irregular due to uneven invasion of the bone in contradistinction to the smooth cystic lesions described above. In the absence of cystic bone lesions and calcification in the soft tissue mass, differentiation between the two conditions may be very difficult and may require biopsy.

In osteoarthritis cystic lesions in subchondral bone are frequently demonstrated but these are relatively less prominent in proportion to the narrowing of the cartilage space, the productive changes and joint deformities.

Cysts of considerable size are frequently seen in rheumatoid arthritis, where we also see synovial swelling of the joints. The swelling is usually smooth and not lobular. Demineralization of bone, joint narrowing, and usually polyarticular involvement dif-

Fig. 4. Case III. Villonodular synovitis of tendon sheath of the little finger, showing moderately severe bone involvement associated with a mildly painful soft tissue mass.

ferentiate this disease roentgenographically from villonodular synovitis. The clinical findings of elevation of the sedimentation rate, heat and redness of the joint and other clinical and laboratory data must all be evaluated.

Tophaceous gout may mimic the roentgenographic appearance of villonodular synovitis with soft tissue swelling, bone erosion and cyst formation, although the clinical signs and symptoms are entirely different. As in other fields of roentgen examination, a knowledge of the patient's clinical findings is most important.

REPORT OF CASES

Case I. A twenty-three year old female noted gradual enlargement of the second toe for approximately one year. The swelling was painless until a few weeks prior to admission and roentgenographic studies at that time showed marked soft tissue swelling surrounding the middle phalanx which contained a cystic lesion (Fig. 1).

At operation a mass was found in the sheath of the long extensor tendon penetrating into the distal interphalangeal joint and into the shaft of the middle phalanx. The toe was amputated. Histologic study of a section of the phalanx showed erosion and invasion of the

Fig. 5. Case iv. Villonodular synovitis of tendon sheath of the little finger, showing severe bone involvement associated with marked soft tissue swelling.

shaft of the bone by granulomatous tissue (Fig. 2). There was evidence of polygonal, ovoid and spindle cells interspersed with numerous multinucleated giant cells. This is the characteristic appearance of villonodular synovitis of tendon sheaths, also known as giant cell tumor or xanthoma of tendon sheaths.

Case ii, iii and iv. The history in each of these 3 cases of villonodular synovitis of a tendon sheath of a finger was essentially the same as in Case i, there being a slowly progressive soft tissue swelling with increasing discomfort. The duration of symptoms was usually one or more years. Roentgenograms (Fig. 3, 4 and 5) showed cystic destructive lesions with normal mineralization of the phalanges, and the massive nodular soft tissue swelling surrounding the areas of bone destruction.

Treatment in all of these cases consisted of excision of the involved tendon sheath and curetting of the bone cysts produced by the invading granulomatous tissue.

Case v. A forty-four year old male described a hard mass on the dorsum of his left wrist which he stated had been present since early childhood. After a fall he developed a red discoloration over this area and the mass gradually increased in size. He had only slight slight pain and no limitation of motion. Roentgen examination showed a cystic rarefaction of the radial styloid with marked soft tissue swelling over the dorsum of the wrist (Fig. 6). The cartilage space of the wrist was of normal width, the mineralization was normal and there were no changes of either rheumatoid or osteoarthritic nature present.

Surgical exploration revealed an encapsulated purplish villous mass protruding from the wrist joint and encompassing the tendons of the extensor pollicis and the abductor pollicis

Fig. 6. Case v. Villonodular synovitis of the tendon sheaths of the extensor pollicis and abductor pollicis longus and of the wrist joint. There is marked soft tissue swelling, subcortical and subarticular bone cysts, and good preservation of the wrist joint space. (Reproduced by permission.[6])

longus. The mass was dissected from the tendon sheaths with difficulty and was seen to extend into the radius. The cystic area of the radius was curetted.

Microscopic examination showed the findings of villonodular synovitis with histiocytes containing cholesterol and hemosiderin, and also many multinucleated giant cells. Epithelioid tubercles without caseation were described. Cultures taken from the lesion showed no growth and guinea pig inoculation, culture and smears for tuberculosis were negative.

Case vi. A fifty-seven year old female first noted occasional aching in her left elbow following trauma in 1951, five years prior to operation. No roentgenograms were made at that time but two years later in 1953 following a fall, roentgen examination showed a multiloculated cystic area in the lateral epicondyle of the humerus. A large nodular intracapsular soft tissue mass was seen anterior to the elbow joint (Fig. 7). Since the initial injury the elbow had remained swollen, she complained of slight but progressive increase of pain and slight diminution of range of motion. Preoperative roentgenograms in 1955 showed findings identical to those of the first examination and a diagnosis of villonodular synovitis was made. A skeletal survey revealed no other lesions, blood chemistry and blood cell counts were normal.

The elbow was explored and a diffuse villous synovitis was found within the capsule extending into the lateral epicondyle. The cystic area was curetted and filled with bone chips, and the affected synovia excised. Microscopic examination showed pigmented villonodular synovitis.

Case vii. A forty-two year old male had pain and swelling of one elbow for a period of six years. Both swelling and pain became progressively worse during this time. This case

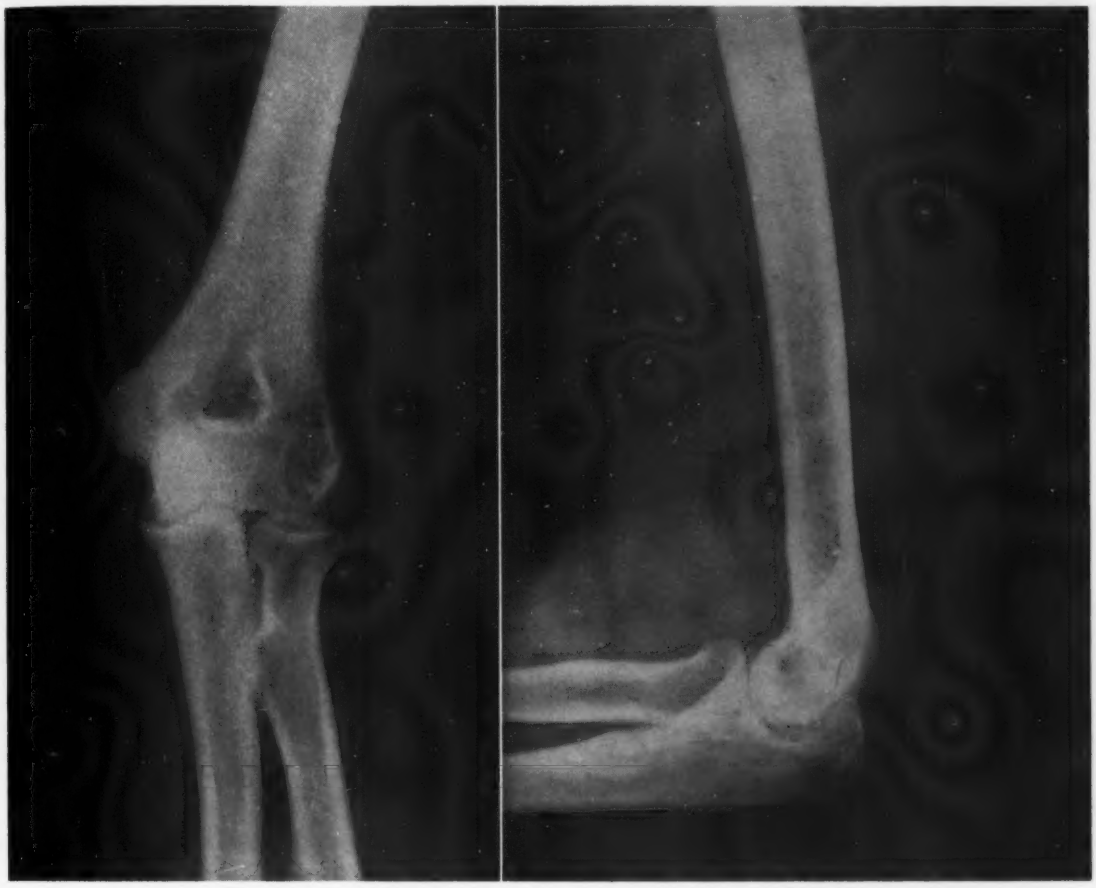

Fig. 7. Case vi. Villonodular synovitis of the elbow. Marked lobular synovial swelling seen anteriorly. Multicystic lesion in the lateral condyle was caused by invading granulomatous tissue.

differs from Case vi only because no history of trauma could be elicited. Roentgen examination showed a large multiloculated cystic lesion on the olecranon process of the ulna and synovial swelling of the elbow joint (Fig. 8).

On exploration a diffuse villous synovitis extending from the elbow joint into the ulna was found, and microscopic examination showed the findings of pigmented villonodular synovitis. Culture and guinea pig inoculation were negative.

Case viii. A thirty-nine year old construction worker noted onset of pain in his right hip, without preceding history of trauma, three years preoperatively. The pain which was mild at first became progressively worse and he complained of stiffness of the hip after sitting or lying down for long periods.

Roentgenographic examination showed multiple cysts within the femoral head and neck and surrounding the acetabulum (Fig. 9). A soft tissue mass could not be demonstrated; however, intracapsular masses or swelling of the hip is always difficult or impossible to demonstrate due to the large amount of muscle tissue which surrounds the hip. The fullness of the obturator muscle, the obturator sign, which may be seen with acute synovitis of the hip is a reaction of the tissue to acute inflammation and would not be present in a chronic process such as villonodular synovitis.

Skeletal survey, blood chemistry studies and physical examination showed no evidence of systemic disease.

At operation the capsule of the hip was bulging and hyperplastic synovial villi protruded through the incised capsule. The gross appearance was that of a diffuse pigmented villonodular synovitis. The villous tissue was seen to extend into the cartilage of the head and acetabulum and into the bone of the femoral neck.

FIG. 8. Case VII. Villonodular synovitis of the elbow with a large multicystic lesion in the olecranon process of the ulna. There is marked lobular synovial swelling, normal mineralization of the bone and normal preservation of the joint space. (Reproduced by permission.[6])

FIG. 9. Case VIII. Villonodular synovitis of the hip with multiple cysts in the femoral head and neck and in the acetabular fossa. The distended capsule found at operation was not visible on the roentgenograms.

Fig. 10. Case IX. Villonodular synovitis of the ankle involving the sheaths of the peroneal tendons. Erosions are seen in the tibia and fibula and small cysts are present in the tibia and talus. Marked soft tissue swelling is present, especially at the lateral aspect of the ankle joint. (Reproduced by permission.[6])

Histologic examination showed typical pigmented villonodular synovitis with cholesterol and hemosiderin containing histiocytes.

Three similar cases have been described previously. Two were in the "Case Histories of the Massachusetts General Hospital of 1944 and 1951."[12] A case with more extensive synovitis was reported recently by Carr, Berley and Davis.[1] A twenty-seven year old male presented with an abdominal mass thought to be a psoas abscess and cystic changes of one hip. The mass was found to be pigmented villonodular synovitis extending from the hip joint into the psoas muscle, forming a retroperitoneal mass. Extracapsular extension is not unusual, and we have observed it in several cases without bone lesions and also in Case II and VI.

Case IX. A thirty-four year old female injured her ankle in 1942, six years prior to operation. Following the injury the ankle remained swollen and moderately painful for the subsequent six years. A history of intermittent

FIG. 11. Case IX. Eight years past partial synovectomy and roentgen therapy. The preoperatively seen bone lesions are still present, but the soft tissue swelling has not recurred and the patient is asymptomatic.

minor injuries and progressive swelling of the ankle was obtained. On admission there was marked swelling of the ankle joint, mainly on the lateral aspect, and no limitation of motion was noted. The swelling was moderately tender.

Roentgenographic examination showed a smooth pressure defect in the fibula and small cystic defects in the tibia and talus with large soft tissue masses anteriorly and laterally (Fig. 10).

At operation a mass was found below the subcutaneous tissue bulging out from the lateral and anterior aspect of the ankle joint. The mass was dark in color and only partially encapsulated, extending into the tendon sheaths of the peroneal tendons, the tibiofibular ligament and into the tibia and talus. It was not possible to excise the mass completely. Histologic examination showed pigmented villonodular synovitis.

Because of the incomplete removal of the synovial masses, a course of roentgen therapy was started ten days postoperataively. A dose of 1,000 r in air to each of two lateral opposing ports was delivered in one week. This, as discussed before, is a high dose for a benign condition.

At present, eight years after treatment, the patient is well and there is no evidence of recurrence. A recent roentgenographic examination shows no change in the preoperatively demonstrated bone lesions but there is absence of soft tissue swelling (Fig. 11).

TREATMENT

The treatment of choice of villonodular synovitis is synovectomy. As much as possible of the diseased synovial tissue should be excised.

Roentgen therapy without surgery has been advocated in the past, but we feel that surgery, aside from producing apparently permanent relief in a great percentage of cases, also has the advantage that it permits definite histologic proof that one is dealing with a benign condition and not a synovial sarcoma. No conclusive information is available as to the recurrence rate since the number of reported cases is relatively small. De Santo and Wilson[2] cite 14 recurrences in 41 reported cases. Most of the cases described here have not been observed long enough postoperatively to determine recurrence rate.

Roentgen therapy has not been uniformly successful in preventing further growth or recurrence and it was found that rather large doses were required, doses much higher than are usually employed in the treatment of benign conditions. Tissue doses of 1,800 to 2,000 r in a period of two weeks have been given at this hospital and similar doses are cited by Shafer and Larmon.[7] At this time we reserve roentgen therapy for postoperative recurrences.

SUMMARY

Bone erosion and formation of uni- and multiloculated cysts may be seen adjacent to the nodular soft tissue masses of villonodular synovitis of tendon sheath and joint synovium. Nine cases are reported and the differential diagnosis is discussed.

The diagnosis of villonodular synovitis with or without bone lesions should be based upon the following criteria: (1) monarticular involvement; (2) normal preservation of the joint space; (3) normal mineralization of juxta-articular bone; (4) nodular intra-articular synovial swelling; (5) lack of calcification within the soft tissue masses; (6) absence of systemic disease; (7) chronicity, with duration of symptoms from several months to several years, with relatively little pain in the affected joint and little if any limitation of motion.

Treatment is by synovectomy. Roentgen therapy is reserved for recurrences.

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SOLITARY PLASMA CELL MYELOMA WITH NEW BONE FORMATION*

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THE roentgenographic findings in plasma cell myeloma are generally uniform, consisting of so-called "punched-out" areas of increased translucency of the involved portion of the bone. The myeloma involves the flat bones of the skull, the ribs, spine and pelvis, as well as the long bones of the extremities. The punched-out areas represent well-circumscribed osteolytic destruction. There may be also a change of the bony structure, characterized by a coarse trabeculation. The most important feature is the complete lack of hypercalcification and new bone formation. This is well-described in the textbook of Roentgen-Diagnostics by Schinz, Baensch, Friedl and Uehlinger,[8] who state: "The single focus usually causes a circumscribed osteolysis without any reactive new bone formation." The same description of the roentgenographic findings is given in many other recent publications. Carson, Ackerman and Maltby[1] for example, state in their article of August, 1955: "The lesion of myeloma is a purely osteolytic one, so that roentgenographic evidence of new bone formation or periosteal reaction would suggest that the process is other than myeloma."

Markovits[4] also emphasizes the lack of reactive new bone formation. He states that periosteal new bone formation is infrequent. No new bone formation is mentioned in any publication in cases where the flat bones are involved.

Rib fractures due to multiple myelomatosis are not infrequent.[9] We have not found any description of callus formation on these rib fractures. In a recent report, however, Russo and Brown[7] described "some recalcification and bone regeneration" in a case of pathologic fracture of a myelomatosis of the first lumbar vertebra. Serial roentgenograms for a period of four years between the fracture and the time of treatment showed decalcification of the involved bone. Surgical and radiation treatment was followed by a very slow recalcification and osteoid regeneration.

Paul and Pohle,[5] in a collection of 45 cases of myeloma, have found only 5 cases with recalcification, but no cases with new bone formation.

The question may be raised whether the different therapeutic methods, which are used for the treatment of myeloma, can produce recalcification and new bone formation. Experience has shown that NH_2, urethane and roentgen therapy can retard growth of the primary lesion or lesions and can delay spread of the process to other locations. With this in mind, one might expect recalcification or reactive new bone formation around the osteolytic lesion, in response to one of these agents. We have found only one case in the literature in which the phenomenon of recalcification and new bone formation has been reported. This is the myeloma case of Rundles and Reeves.[6] They observed recalcification of the fractured distal clavicle five months after beginning of urethane therapy. If we agree with the authors, as seems reasonable, that the new bone formation in their case was a result of a specific therapy, there remains only one case in which true recalcification and new bone formation in the absence of any specific treatment has been reported. This is the case of Krainin, D'Angio and Smelin.[3] These authors report a case of multiple myeloma with typical punched-out areas in all bones involved. Significant and different were the findings in the left humerus. Here they found a diffuse *productive* process involving the entire shaft. Dense bone spicules were arranged at right angles to the shaft proper. The au-

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Fig. 1. (*A* and *B*) Dorsal spine, anteroposterior and lateral roentgenograms, at the time of admission, November 11, 1952. Round, well defined osteolytic areas of the bodies, pedicles and laminae of the eighth, ninth and tenth dorsal vertebrae. Also evidence of some hypercalcification and new bone formation, especially at the right side and the anterior aspect of the bodies of these vertebrae. Narrowing of the interspace between the eighth and ninth dorsal vertebrae.

thors state that they could find in the literature no other case in which a significant degree of new bone formation was visualized, either on histologic examination or on the roentgenogram aside from those at the site of pathologic fractures.

We feel justified in publishing the following case of solitary plasma cell myeloma, which showed, from the beginning, in the presence of the typical osteolytic changes *a high degree of hypercalcification and new bone formation*. This case, besides that described by Krainin, D'Angio and Smelin, is the only other instance of hypercalcifica-

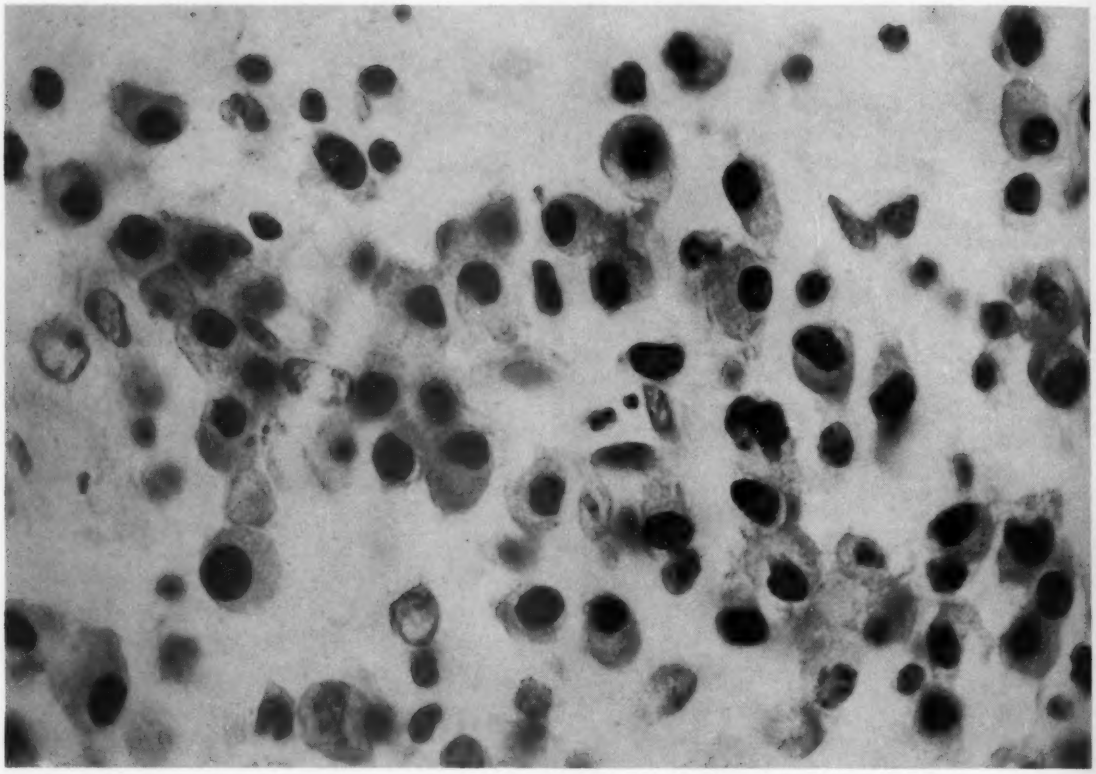

Fig. 2. Photomicrograph, showing a typical section of the tumor with numerous darkly stained plasma cells (1098×).

tion and new bone formation in myeloma, observed before any treatment.

REPORT OF A CASE

A forty-eight-year old white male laborer entered the hospital on November 10, 1952 because of progressive difficulty in walking since August, 1952, and left hypochondric pain since May, 1952. By the time of admission he had weakness and loss of sensation in both legs and hips, sexual impotence, and difficulty in starting his urinary stream. In 1951 the patient had had a penile sore, followed by skin lesions over the entire body, with persistently negative blood tests for syphilis. He received 8,000,000 units of penicillin, then developed angioneurotic edema of the body. The treatment was discontinued and replaced by weekly bismuth injections for four months. The patient reported heavy alcohol consumption during the past twenty years.

Physical examination revealed small, discrete nodules in the skin of the abdominal wall, marked distention of the abdomen and a liver edge palpable 4 fingers below the costal margin. Neurologic examination showed evidence of multiple long tract spinal involvement below the area of the eighth thoracic dermatome bilaterally, including both motor and sensory changes, with slight sacral sparing. Serologic tests for syphilis were negative, and blood proteins and sedimentation rate were normal. A lumbar puncture on November 13, 1952 revealed a complete block, grossly bloody spinal fluid with yellow supernatant fluid and total protein of 3.2 gm. per cent. Roentgenograms of the dorsal spine (Fig. 1*A* and *B*) showed destructive lesions with some proliferation at D-8, 9 and 10. Complete bone survey was otherwise normal. Myelography on November 13, 1952 showed evidence of a mass extending from L-1 up to a complete block at D-10.

Laminectomy from D-9 through D-12 was performed by Doctor Murton R. Shaver* on November 19, 1952, with removal of extramedullary tissue. Sections of the tumor tissue were reported by Dr. Alfred Plaut† to show a

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highly cellular neoplasm, with typical "wheel-spoke" patterns of chromatin in many of the cells (Fig. 2). A diagnosis of myeloma was made. The patient regained much of his neurologic functioning after operation, and was given several courses of deep roentgen therapy at intervals over the next year and a half. From 1954 to 1956 there was breakdown of the skin over D-9 to D-11 and a deep ulcer, down to the muscle layer, formed and progressed in size until May, 1956. The ulcer has been stable, with no improvement in it since that time. The patient has been maintained at bedrest because of the marked changes in his lower spine which contraindicate weight bearing in that area. A sternal marrow smear in January, 1953, and repeated studies of the serum proteins have remained normal up to this time. Urine specimens have been consistently negative for Bence Jones protein.

Fig. 3. Dorsal spine, lateral roentgenogram, on November 21, 1952, one day after laminectomy. Osteolytic changes as well as new bone formation of the eighth, ninth and tenth dorsal vertebrae are well visualized.

Fig. 4. Dorsal spine, oblique roentgenogram, nine days after laminectomy. The luxuriant formation of osteoid tissue, crossing the interspace between the eighth and ninth dorsal vertebrae is well visualized.

At present time, May, 1957, the patient is confined at bedrest, but remains alert and has had no change in his neurologic status since his original postoperative improvement in 1952.

Repeated roentgenographic bone surveys have shown no evidence of any bone lesions beyond those described in the dorsal and lumbar

Fig. 5. (*A* and *B*) Dorsal spine, anteroposterior and lateral roentgenogram, on October 6, 1955, three years after admission and laminectomy. Marked diminution of the osteolytic changes. Regeneration of the involved vertebrae. Marked osteosclerosis and extensive new bone formation. Narrowing and deformity of the body of the eleventh dorsal vertebra due to osteoporosis.

spine. Figures 1, 3, 4, 5 and 6 illustrate the remarkable amount of reactive new bone formation which has occurred. Roentgenograms of the spine made on October 3, 1956 (Fig. 7, *A* and *B*) show regeneration of the bony structure of the vertebral bodies D-8, D-9 and D-10. The osteolytic destruction has nearly completely disappeared, and instead there is a high grade of hypercalcification and a luxuriant formation of sclerotic osteoid tissue in and about D-8 and D-9. Recent roentgenograms on April 18, 1957 show further progress of new bone formation. No osteolytic changes of the lower dorsal and upper lumbar spine are visualized.

Comment. A case of solitary plasma cell myeloma has been described. The case shows several features which are unusual.

Roentgenograms of the dorsal spine on the patient's admission showed round areas of increased translucency as well as partial hypercalcification and new bone formation

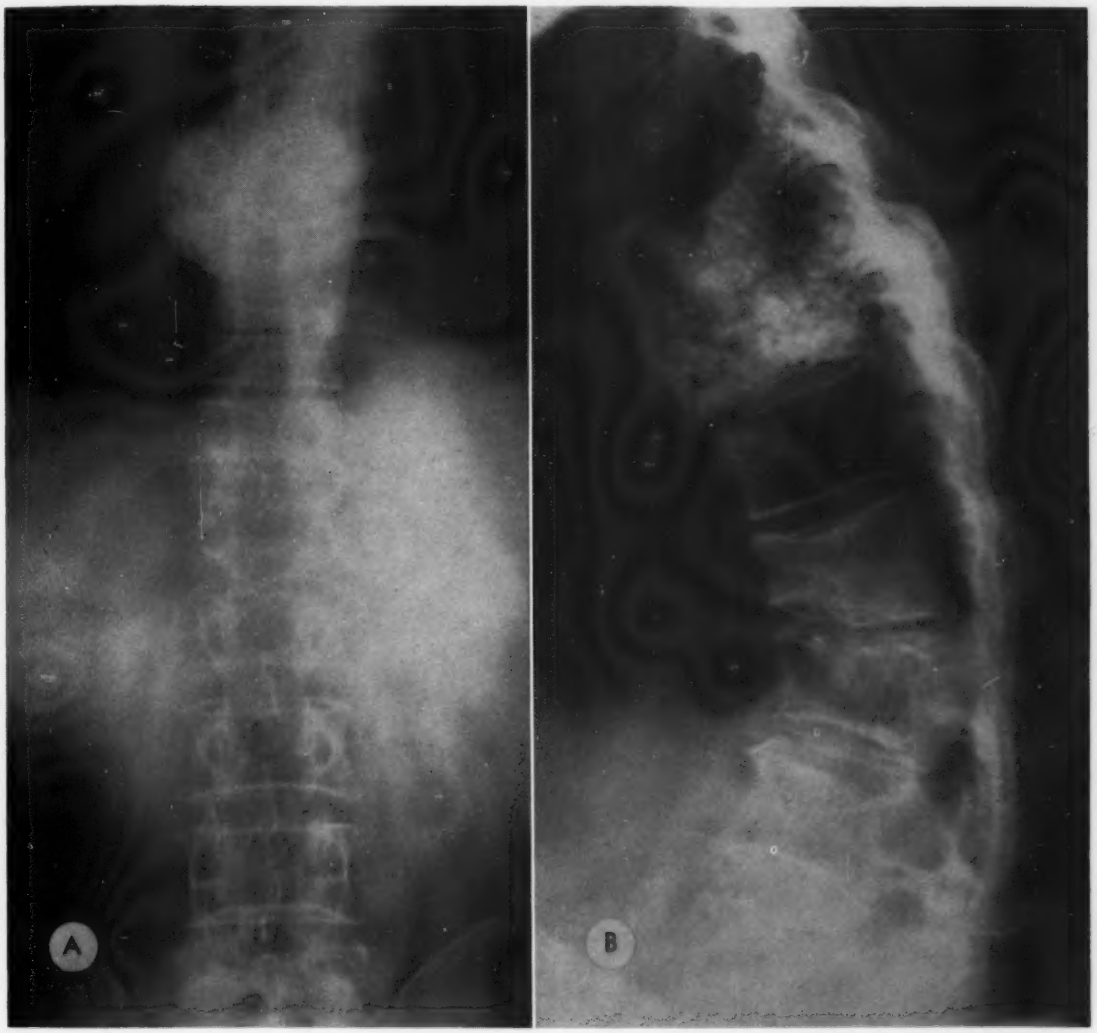

Fig. 6. (*A* and *B*) Dorsal and lumbar spine, anteroposterior and lateral roentgenograms, on January 30, 1956. The eighth, ninth and tenth dorsal vertebrae show further regeneration. Osteoporosis of the eleventh dorsal vertebra unchanged. New osteoporotic involvement of the twelfth dorsal vertebra.

of the eighth, ninth and tenth dorsal vertebrae. These were diagnosed as osteolytic and osteoblastic metastases. Histologic examination of the surgical specimen revealed plasma cell myeloma. Extension of the pathologic process across the intervertebral disks and involvement of contiguous vertebrae, as visualized on the roentgenograms, is unusual in plasma cell myeloma. In the discussion following the paper by Paul and Pohle,[5] however, Paul emphasizes: "I think an important feature is that this type of extension whether it occurs around the margins or through the disk, is certainly more commonly seen in myeloma than in metastatic carcinoma."

As mentioned above, original new bone formation in plasma cell myeloma has been observed and published only once in the literature.[3] One other case with new bone formation was observed, after treatment with urethane.[6] The question arises whether in our case the new bone formation is to be attributed to any therapeutic agent. Our patient received antisyphilitic treatment with bismuth and penicillin a few months

Fig. 7. (*A* and *B*) Dorsal spine, oblique and lateral roentgenograms, on October 3, 1956, four years after admission. Roentgenographic findings generally the same as in January. No further progress of the osteoporosis of the eleventh and twelfth dorsal vertebrae. Roentgenograms of the cervical and lumbar spine taken on the same date were negative. The skeletal survey and chest examination were also negative.

before admission. Although the treatment with both drugs was of short duration, we cannot deny the possibility of a relationship between this treatment and the new bone formation.

A second question is whether there exists any relationship between this unusual myeloma lesion and a syphilitic process. Under our observation, repeated studies have shown no evidence to support a diagnosis of any syphilitic process. Blood tests for syphilis have always been negative and the 2 Kolmer units found in the spinal fluid are well within the limits of false positive.

Such false positive findings in the spinal fluid are common in the presence of tumors of the central nervous system. Therefore, we can deny, in our patient, the presence of a syphilitic process. Also, a change of the pathologic findings in myeloma by a concomitant syphilis has never been reported.

A third question which might be raised is whether or not this case represents one of "solitary myeloma." In defining this problem, we should like to cite the work of Bichel and Kirketerp as summarized by Snapper *et al.*[10] in their book on myeloma: "They laid down rigid criteria for the diagnosis and accepted only two cases as proven. Before considering the diagnosis of solitary myeloma, its presence should be established by biopsy. A skeletal survey should eliminate the presence of other radiologic lesions. The sternal marrow puncture should be negative. As a rule, hyperglobulinemia and Bence-Jones proteinuria should be absent. . . . Bence-Jones proteinuria has been described in cases of solitary myeloma which were confirmed at autopsy (reference cited). This must be a rare occurrence and in our material the appearance of Bence-Jones proteinuria is a prognostic sign predicting early spread of the disease. Finally, no generalization of the myelomatous process should occur during a long period of observation." Snapper *et al.* cite the acceptance of these criteria by numerous authors. In discussing the report of Christopherson and Miller[2] in 1950, time criteria are mentioned as " . . . cases which had been followed for at least three years without signs of generalized dissemination . . . an observation of less than three years is too short to exclude the possibility of future dissemination of the disease process." Attention to each one of these criteria shows that our case fulfills them all. Thus we feel at the present time that our case fits into the clinical category of solitary myeloma.

SUMMARY

A case of solitary plasma cell myeloma, studied for over four years, has been presented, with the very unusual feature of new bone formation, recalcification and regeneration. This is the second reported case of plasma cell myeloma with new bone formation, and the first case in which a flat bone is involved. Such cases represent very rare exceptions to the rule that new bone formation excludes the diagnosis of myeloma.

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LYMPHOSARCOMA SHOWING UNUSUAL BONE MANIFESTATIONS*

A CASE REPORT

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THERE are several reports of Hodgkin's disease in which the tumor initially manifested itself in bone.[1,4,8] However, we have been able to find only 3 cases of lymphosarcoma in which the bone lesion was the first and only lesion discernible at the time of diagnosis.[2,10,12] We would like to add our case to this small group, especially since in our case the bone lesions were quite unusual in their distribution.

REPORT OF A CASE

A fifty year old white male janitor was admitted to the Syracuse Veterans Administration Hospital in June of 1955 complaining of pain and swelling in the middle phalanx of the fifth finger of his right hand for six months. The patient had no complaints of a general nature other than asthma of many years' duration.

The past history, family history and system review were all noncontributory.

Physical examination revealed a well developed, thin white male whose vital signs were within normal limits. The chest was moderately emphysematous. There was a soft, slightly tender, fusiform swelling of the middle phalanx of the fifth finger of the right hand with normal color and temperature of the overlying skin.

All laboratory tests were within normal limits.

Roentgenographic examination of the right hand (Fig. 1) revealed a destructive lesion in the middle phalanx of the fifth finger, probably centered in the diaphysis. The lesion had broken through the cortex and a soft tissue mass was present. The bone appeared to be slightly expanded and there was a suggestion of trabeculation. It was the radiologist's impression that the lesion most likely represented a giant cell tumor of the bone. Amputation of the fifth finger at the metacarpal-phalangeal joint was performed and the pathologist's diagnosis was large cell lymphosarcoma.* The gross and microscopic appearance of the tumor is illustrated in Figures 2 and 3.

FIG. 1. Roentgenogram showing destructive lesion in middle phalanx of fifth finger with associated soft tissue mass. Some expansion of bone best seen on radial aspects.

* We wish to thank Dr. J. Albert Schaefer, Chief, Laboratory Service, Veterans Administration Hospital, Syracuse, New York, Assistant Professor of Pathology, State University of New York College of Medicine at Syracuse for the original diagnosis. This material has been reviewed by Dr. J. Howard Ferguson, Professor and Chairman, Department of Pathology, State University of New York College of Medicine at Syracuse, and also by members of The Armed Forces Institute of Pathology. We wish to thank them for their assistance.

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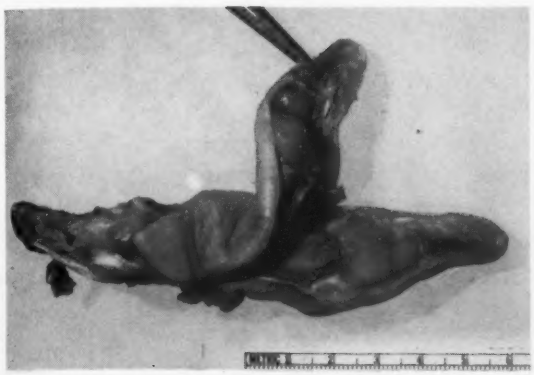

Fig. 2. Longitudinal cross-section of the gross specimen of the tumor replaced phalanx.

A roentgenogram of the chest taken prior to surgery showed no enlargement of mediastinal lymph nodes. A roentgenographic survey of the bones failed to show any other lesions. Bone marrow aspiration was within normal limits with no evidence of malignant cells.

The patient was discharged on July 29, 1955, and remained in good health until March, 1956 when he noticed pain and slight swelling in his right ankle. He reported to the Orthopedic Clinic where roentgenograms revealed destructive lesions in both ankles (Fig. 4), and he was readmitted in March of 1956 for further investigation.

Physical examination again revealed a well developed, thin white male in no distress who did not appear chronically ill. The relevant findings were minimal swelling and slight tenderness of the anterior aspect and medial maleolus of the right ankle. There was surgical absence of the fifth finger of the right hand. No lymph node enlargement was noted and no organs were palpable in the abdomen.

The hematocrit was 49 and the white blood cell count 9,200 with a normal differential. The fasting blood sugar was 91 mg. per cent, the nonprotein nitrogen 45 mg. per cent, calcium 10.2 mg. per cent, phosphorus 5.0 mg. per cent, alkaline phosphatase 2.3 Bodansky units and acid phosphatase 0.64 Bessey Lowry units. A bone marrow aspiration revealed a normal marrow with a slight increase in lymphocytes.

Roentgenographic examination of the ankles revealed multiple lytic lesions in the tibiae and fibulae of both lower extremities as illustrated in Figure 4. These lesions were located primarily in the cortex and no soft tissue mass could be seen. The largest lesion measured 2.5

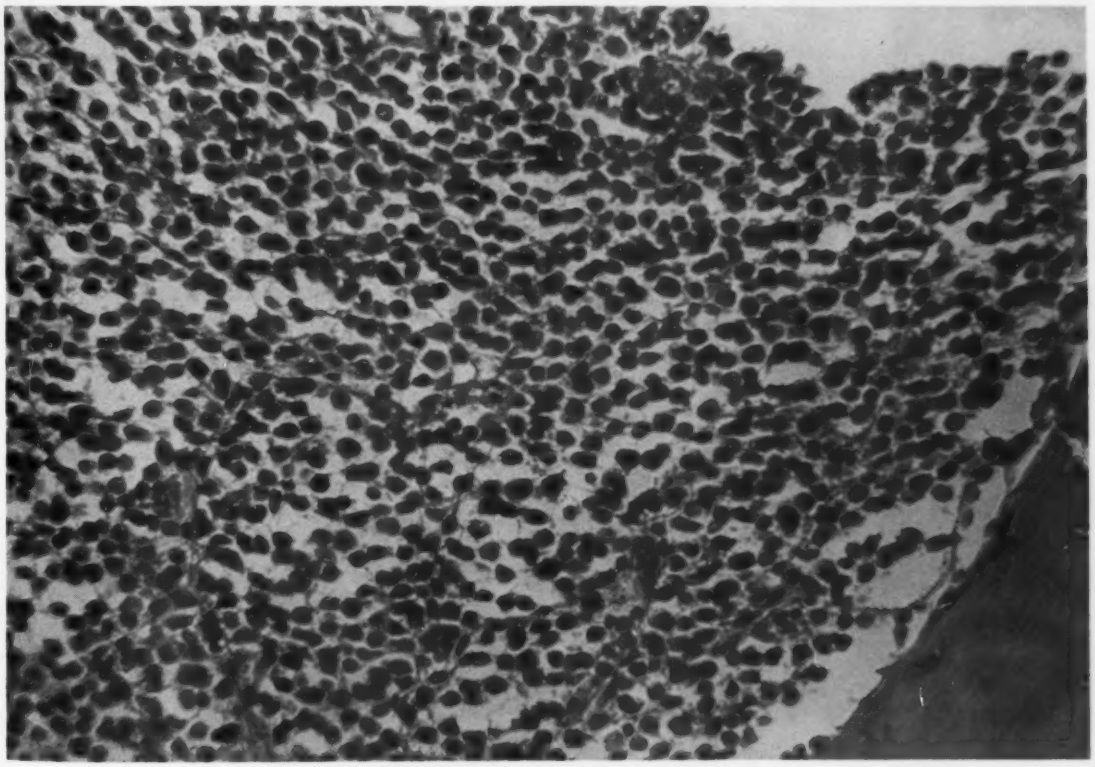

Fig. 3. Photomicrograph showing the histologic pattern of the phalangeal lymphosarcoma (×177).

cm. in greatest diameter and was located in the right tibia. It was slightly irregular along its margin while the smaller lesions have had smooth margins. No periosteal reaction was noted.

Complete skeletal survey showed lesions similar to those seen in the ankles, in the forearms and the hands, as seen in Figures 5 and 6. The skull, thoracic cage, spine, and pelvis were within normal limits.

Fig. 4. Roentgenograms of the ankles showing destructive lesions in the right lower tibia and the left lower fibula.

A gastrointestinal series and an intravenous pyelogram were normal. Biopsy of the lesion in the right tibia presented the same histologic picture as that of the lesion of the fifth finger; namely, large cell lymphosarcoma. The patient was given a course of nitrogen mustard therapy and discharged for follow-up by the hematologist. When seen in May, 1956, the patient was in good general health.

Comment. This case exhibits several unusual aspects. It represents an unusual bone lesion for lymphosarcoma and an unusual type of pathology for an osteolytic lesion of the cortex of a long bone. As mentioned in the introduction, there are few authenticated case reports in which lymphosarcoma presents with a bone lesion as its initial manifestation. When lymphosarcoma does involve the skeletal system, it is usually restricted to the bones of the trunk or proximal portion of the extremities.[2,11,12] In our review of the literature we have failed to find mention of involvement of the hands or feet.

In our patient not only was the initial lesion located very peripherally, but all subsequent lesions showed a peculiar predilection for the small bones of the hands and the feet as well as the distal portion of the forearms and lower legs.

Vieta, Friedell and Craver[12] have re-

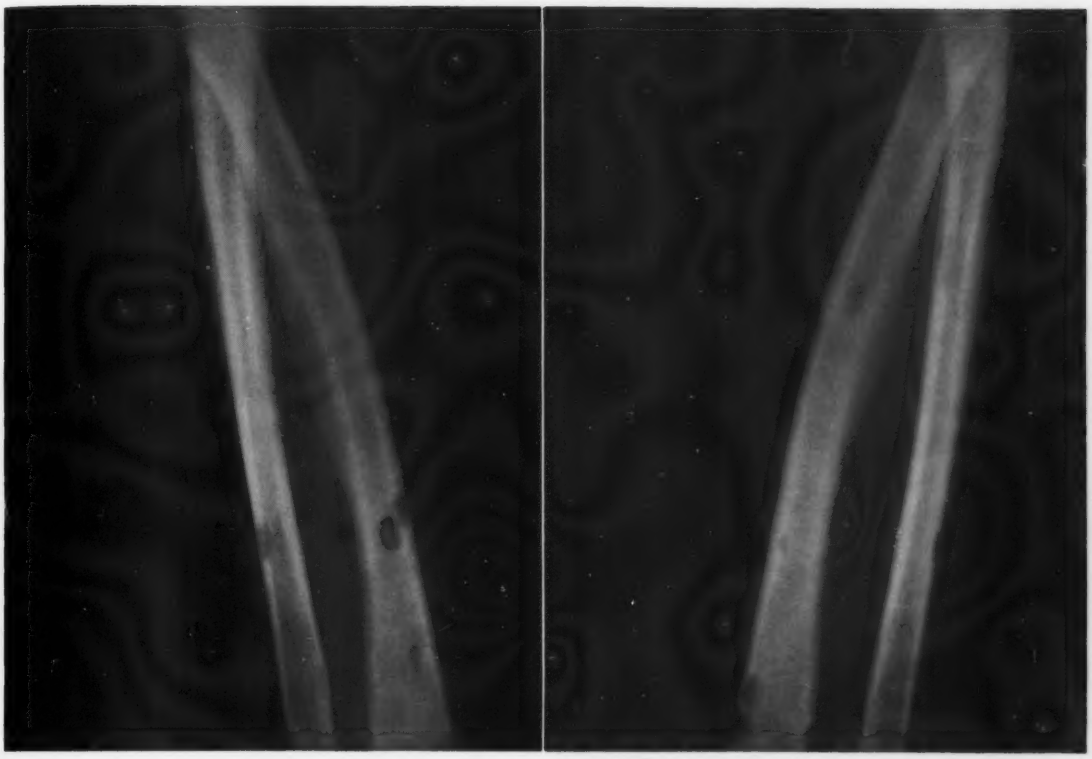

FIG. 5. Multiple osteolytic lesions in both forearms.

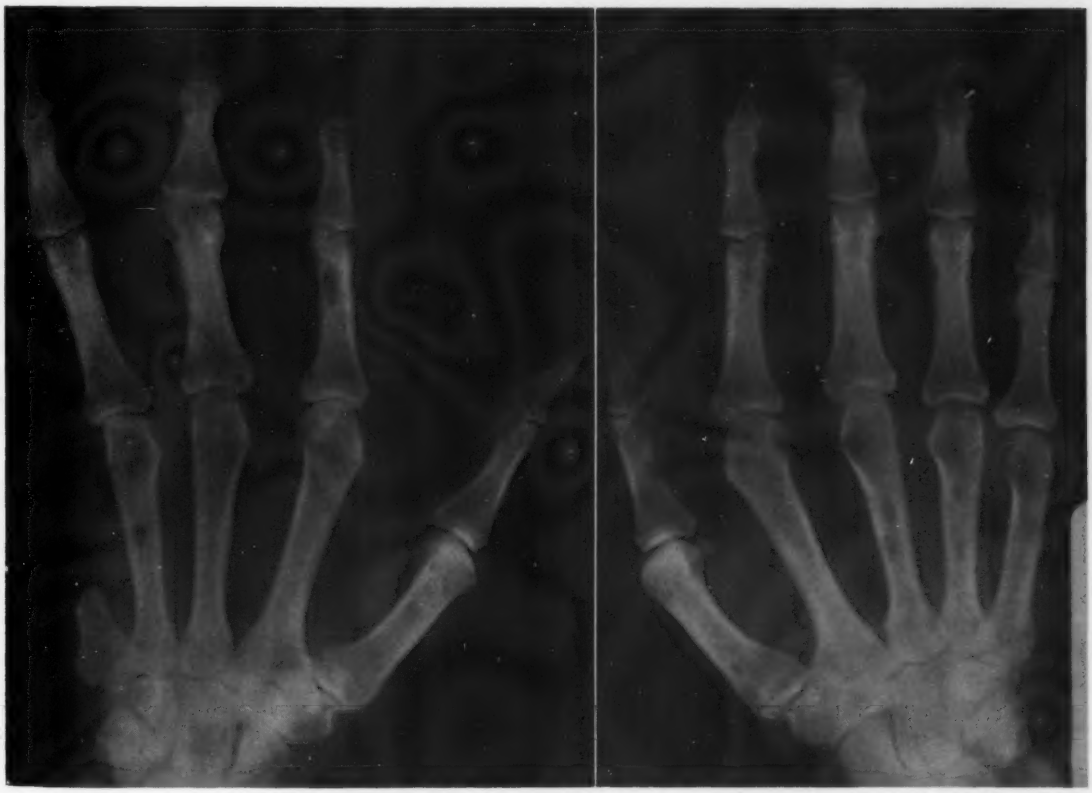

FIG. 6. Roentgenograms of the hands show destructive lesions in phalanges and metacarpals. There is also evidence of degenerative arthritis and there is amputation of left fifth finger.

ported a similar case. In their case the initial lesion was in the skull and was treated with irradiation with apparent excellent results. A lesion histologically identical was discovered in the femur six years later.

The usual bone lesions of lymphosarcoma are medullary in origin. The bone lesions in our patient were in the cortical region of the bones affected. On gross inspection at the time of biopsy of the right ankle, the tumor mass was found to involve the periosteum, cortex of the bone, and the soft tissue immediately overlying the lesion. Such a picture suggests a periosteal origin of the lesion.

The differential diagnosis presented by this case in its later stages would include such diseases as eosinophilic granuloma, multiple myeloma, and metastatic carcinoma.

The distribution of lesions is unusual for eosinophilic granuloma, for the skull and mandible are not involved though the disease is widespread. In contradistinction to the sharp margins of the lesion of eosinophilic granuloma, the larger lesions in our case were irregular.

Multiple "punched out" lesions of the bone occur in multiple myeloma. In addition, there are changes in the architecture of the bone such as osteopetrosis and loss of the finer trabeculation. In widespread myeloma, the skull and spine are nearly always involved.

The lack of appearance of a primary lesion in a patient followed for one year militates against carcinoma with metastases. While single lesions of the small bones of the extremities are known to occur in carcinomas, particularly those primary in the thyroid, lungs or kidneys, such widespread distribution in all extremities would be very unusual.

SUMMARY

Lymphosarcoma, which initially manifests itself as a local bone lesion, is a rare tumor. The wide distribution of the lesions in the bones of the extremities and the predilection to cortical destruction are unusual aspects.

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ADDENDUM

The patient has been followed in our Follow-up Clinic for about nine months since therapy with nitrogen mustard. There has been recalcification of some lesions, but others have appeared. He has now small rubbery axillary lymph nodes. No enlargement of other lymph nodes could be found.

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VOL. 79, NO. 4

METASTATIC CARCINOMA IN THE BONES OF THE HAND*

A REPORT OF TWO CASES

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METASTATIC tumors in the bones of the hand are extremely rare, and few cases have been reported. Coley and Higinbotham[2] mentioned only 2 instances in their report of 47 tumors in the bones of the hands and feet. Of 171 malignant tumors of the hands, Pack[3] likewise noted only 2 of metastatic origin. Bell and Mason[1] collected 12 patients with metastatic tumors of the hand from the literature, and reported 2 additional cases of their own. In 10 of these 14 patients, the primary source was carcinoma of the lung. We have recently seen 2 interesting cases of this type which appear to merit a brief report.

REPORT OF CASES

CASE I. This fifty year old white male entered the hospital in January, 1953 because of swelling and pain in the left thumb of six months' duration. Roentgenograms of the left hand showed an almost complete absence of the first metacarpal bone (Fig. 1) and a biopsy specimen was reported as epidermoid carcinoma. A chest roentgenogram revealed a small nodular density in the right apex, and a clinical diagnosis of bronchogenic carcinoma was considered. The patient was not referred for roentgen therapy at this time. After an amputation through the left forearm, the patient remained well for eight months. He returned to the hospital because of pain and limitation of motion in the right shoulder. Chest roentgenograms at that time revealed a large superior sulcus lesion with lytic involvement of the right first rib. The patient was then referred for roentgen therapy and this lesion received a tumor dose of 5,800 r in thirty-eight days using one million volt radiation. The pain was relieved, and the range of motion of the right arm was markedly improved.

CASE II. This fifty-eight year old white male entered the hospital in July, 1956 because of cough and progressive weight loss of six months' duration. His chest roentgenogram showed a hilar mass with atelectasis of the left lower lobe. An enlarged supraclavicular lymph node was biopsied and reported as epidermoid carcinoma. Roentgenograms of the left hand revealed a lytic lesion in the proximal phalanx of the thumb (Fig. 2) and a needle biopsy in this area showed epidermoid carcinoma. The patient received palliative roentgen therapy to widespread symptomatic metastatic foci. The lesion

FIG. 1. Destructive lesion of the first metacarpal bone of the left hand, metastatic from a silent bronchogenic carcinoma in a fifty year old white male. Pain in the hand was the patient's first complaint.

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FIG. 2. Destructive lesion in the proximal phalanx of the thumb, metastatic from a bronchogenic carcinoma in a fifty-eight year old white male.

in the thumb, however, gave rise to no pain or discomfort and was not treated.

CONCLUSION

Of more than 800 patients with bronchogenic carcinoma seen and treated in our Radiation Clinic, only two have shown metastatic lesions in the bones of the hand, one in a metacarpal bone and the other in a proximal phalanx. Both foci were lytic in nature. It is of interest that the majority of metastatic lesions in the hand arise from primary bronchial cancer and that one of our patients presented the metastatis as his first complaint.

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VOL. 79, NO. 4

EPIDERMOID (EPITHELIAL) CYST OF THE HAND

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EPIDERMOID cyst of the hand is a well-documented, distinct clinical and pathologic entity of rather rare occurrence, judging from the paucity of cases reported in the literature. Perhaps the lesion is not as rare as would be expected from the few reported cases. The authors hope to call more attention to this entity with a case report. Kelly and Clifford[3] reported 4 cases seen within a period of eighteen months. Unnecessarily radical therapeutic measures may be avoided if the correct preoperative diagnosis is made by the radiologist. That radical therapeutic measures have been undertaken is documented in the literature by Carroll's[1] excellent case reports and Dr. M. L. Mason's comment on that paper. It is hoped that the correct preoperative diagnosis may be made more readily with increasing awareness of the existence of the lesion.

ETIOLOGY

Two etiologic theories have been suggested.[6] One hypothesizes the embryologic misplacement of epidermal cells which continue to grow throughout life with or without the stimulus of trauma. The other theory, which the authors feel is more likely, is that the epidermis is displaced within the osseous structures of the affected phalanx as a result of trauma. Once displaced within the phalangeal bone, the epidermis continues to grow, destroying the integrity of the osseous components in its path until surgical intervention removes or destroys all remnants of viable displaced epithelium.

PATHOLOGY

The lesion consists of a cyst the walls of which are made of stratified squamous epithelium. The central portion of the cyst contains horny debris which grossly appears to be white "caseous" material.

HISTORY, SIGNS AND SYMPTOMS

The patient's most frequent complaint is pain over the involved phalanx. Interrogation of the patient in a majority of instances will bring forth a history of antecedent trauma. The initiating traumatic event has occurred from one to thirty-five years before symptoms caused the patient to seek medical advice.[4] Although the pain of which the patient complains is only of a mild nature, it is aggravated by such minor events as grasping objects with the affected digit. The aggravation of pain is demonstrated by the palpation of the attending physician. Usually, the patient will have noticed some increase in size of the affected phalanx. Comparison with the same member of the opposite hand will reveal this to be true. Occasionally, there will be associated erythema of the terminal phalanx. No limitation of motion is present.

ROENTGENOGRAPHIC FINDINGS

Roentgenographic examination of the digit reveals a destructive lesion of the distal phalanx. The lesion is roughly spherical and homogeneous except for the occasional appearance of vacuolated areas within the cyst. The roughly spherical shape is brought out even more strikingly on the roentgengogram by its location, *i.e.*, the terminal phalanx. Sonntag[5] reported a case in the middle phalanx; however, the terminal phalanx had been amputated in his patient at the time of the initiating injury. Frequently, a pathologic fracture is present and the cortical bone may be interrupted on one or more surfaces by the growth of the cyst. No periosteal reaction is present. A thin rim of increased density, representing

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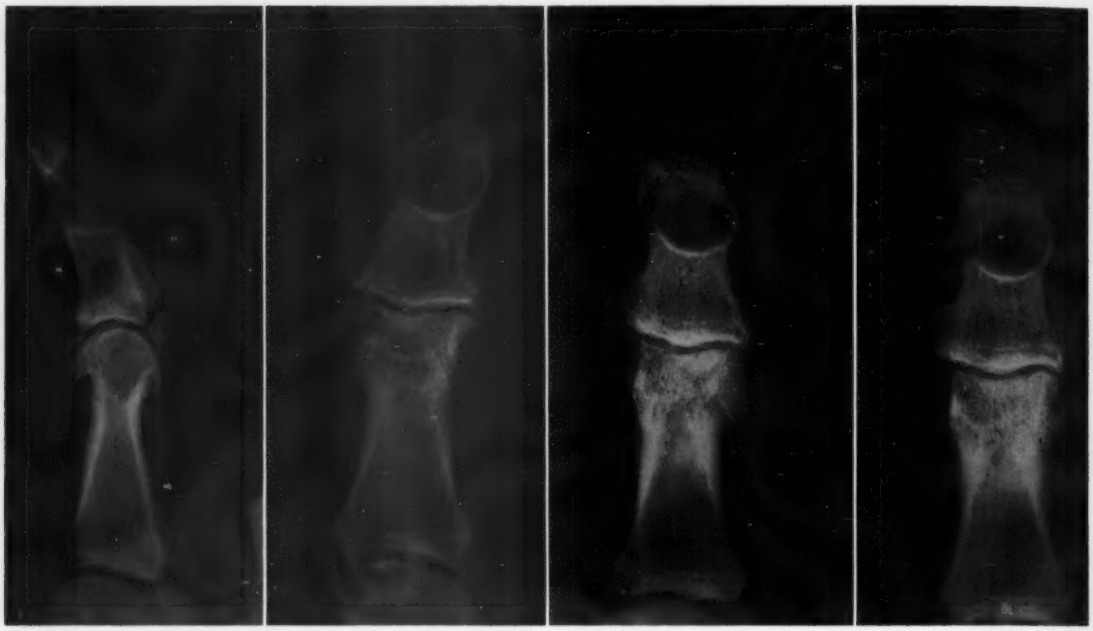

Fig. 1. Roentgenograms of the right thumb in several positions showing the typically spherical cystic lesion of the terminal phalanx with cortical erosion.

condensation of the spongiosa, surrounds the cyst where the cortex has not been broken through. This suggests the benignity of the lesion and lack of true invasion of the surrounding bone.

DIAGNOSIS

The diagnosis seems relatively simple if the previously described features are present and the physician is aware of the condition. The differential diagnosis should include enchondroma, simple bone cyst, giant cell tumor and metastatic malignant tumors.[2] Of these lesions, only enchondroma may cause difficulty but a careful clinical history and roentgenographic examination will most often eliminate it from the list of diseases to be considered.

REPORT OF A CASE

The patient is a sixty-seven year old male who entered the hospital in September, 1956, because of a painful, swollen right thumb. Upon further questioning, he recalled that twelve years prior to the present illness he had sustained an injury to his thumb when it was caught between a truck and a ten-inch pipe. The right thumb was lacerated and the nail sloughed a few weeks later. He was well until two months prior to admission at which time he noticed swelling and pain in the distal phalanx of his right thumb. The pain was mild and was not relieved by hot soaks. A marked increase in tenderness was noted when he squeezed his thumb. Physical examination revealed a swollen, tender distal phalanx of the right thumb. The tenderness increased with palpation and the skin of the volar aspect was erythematous. No limitation of motion was present. The roentgenographic examination (Fig. 1) showed a spherical, well-circumscribed, radiolucent osseous defect measuring approximately 1.0 cm. in diameter in the distal one third of the terminal phalanx of the right thumb. The cortex of the bone was broken through on the volar and dorsal aspects and minimal bony sclerosis surrounded the lesion where it was bounded by spongious bone. A vacuolated appearance was present within the cystic area. Soft tissue swelling was noted on the volar aspect of the thumb. The diagnosis of epidermoid cyst of the distal phalanx was made. The patient was subjected to surgery and the cyst was opened through a lateral incision. The white, fairly thin capsule was opened and "caseous" material was noted within. The capsule was excised and the cyst bed curetted. The pathologic report confirmed the diagnosis of epidermoid cyst of the hand.

DISCUSSION

Epidermoid cyst of the hand is a comparatively rare lesion if the paucity of literature pertaining to it is any indication of its incidence. The diagnosis is not difficult if one is aware of the pathologic entity. Radical therapeutic measures may be averted if a correct diagnosis is made preoperatively. Only by increased awareness of the existence of this lesion can this be made possible. Satisfactory results have followed treatment consisting of removal of the cyst and curettement and cauterization of the cyst bed.[3,4] When this therapy is not feasible because of size and location of the cyst, amputation may be necessary.

CONCLUSION

1. Typical features of epidermoid cyst of the hand are discussed.

2. A case report with typical findings is presented.

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OSTEOGENIC SARCOMA OF THE SKULL ARISING IN PAGET'S DISEASE*

A CASE REPORT

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ALTHOUGH osteogenic sarcoma in Paget's disease is not infrequently encountered, involvement of the skull is seldom noted. The purpose of this report is to cite such an instance. The usually accepted incidence of malignant transformation in Paget's disease is 10 to 15 per cent (Schmorl,[5] Lichtenstein[3]). Coley[1] has stated that about 60 per cent of osteogenic sarcomas in the tibiae arise in pre-existing Paget's disease, and 100 per cent of osteogenic sarcomas in the skull are on the basis of malignant transformation of osteitis deformans. In a similar vein, Sosman in a communication to Sear[6] has stated that osteogenic sarcoma is not found in patients over fifty unless it is on the basis of transition to malignancy in Paget's disease.

Lichtenstein[3] has reported a case of osteogenic sarcoma in the skull originating in Paget's disease, with no other site of involvement of the skeletal system by the pagetoid process. He has pointed out that transition need not be to osteogenic sarcoma, and that malignant change into fibrosarcoma or an anaplastic lesion resembling a malignant giant cell tumor may occur. Chondrosarcoma has also been observed in Paget's disease. At Memorial Hospital in New York, of 23 malignant transformations of Paget's disease, only 3 were in the calvarium, all of osteolytic variety.[7]

It is of interest to note that Sir James Paget's[4] first patient to be reported with bone changes of osteitits deformans was followed from 1852 to 1867 and died of neoplastic disease. At that time, tumor nodules were noted in the pleura, spinal cord and the skull. Of 8 patients of Paget's original series who were followed adequately, 5 developed malignant neoplasms, but only 2 had malignant metaplasia of Paget's disease.

In osteogenic sarcoma arising in Paget's disease, the prognosis is uniformly hopeless. Coley and Sharp,[2] were unable to find a single instance of five year survival.

Paget[4] was impressed with the susceptibility to malignant change and he mentioned this in his original work. He stated in 1888 "It is as if by some gradual general change the osteitis made a patient very liable to cancer or sarcoma, for it has in every case existed many years before the malignant growth appeared, and the liability is general rather than local for the morbid growths do not form exclusively or even chiefly in the diseased bones."

In the present case report it is of additional interest that the patient had a pituitary tumor operated upon twenty years earlier, and which was present at the time of death.

REPORT OF A CASE

The patient was a sixty-one year old white male admitted in a comatose state of one day's duration. There was a past history of an operative procedure involving the pituitary in 1937. At that time, Paget's disease of the skull had been reported.

In June of 1955 (one year prior to admission), a lump in the left temporo-occipital area of the scalp was noted. It was excised for biopsy and the diagnosis of "neoplasm" arising in Paget's disease was made. In November, 1955, the patient received radiation therapy to the soft tissue mass of the skull. The factors were: 250 kv., 2 mm. Cu half-value layer, a 12×7 cm. field in the parietal-occipital region and opposed 14×14 cm. lateral fields at 50 cm. target skin distance. A tumor dose of 4,700 r was delivered in thirty-six days. There was definite re-

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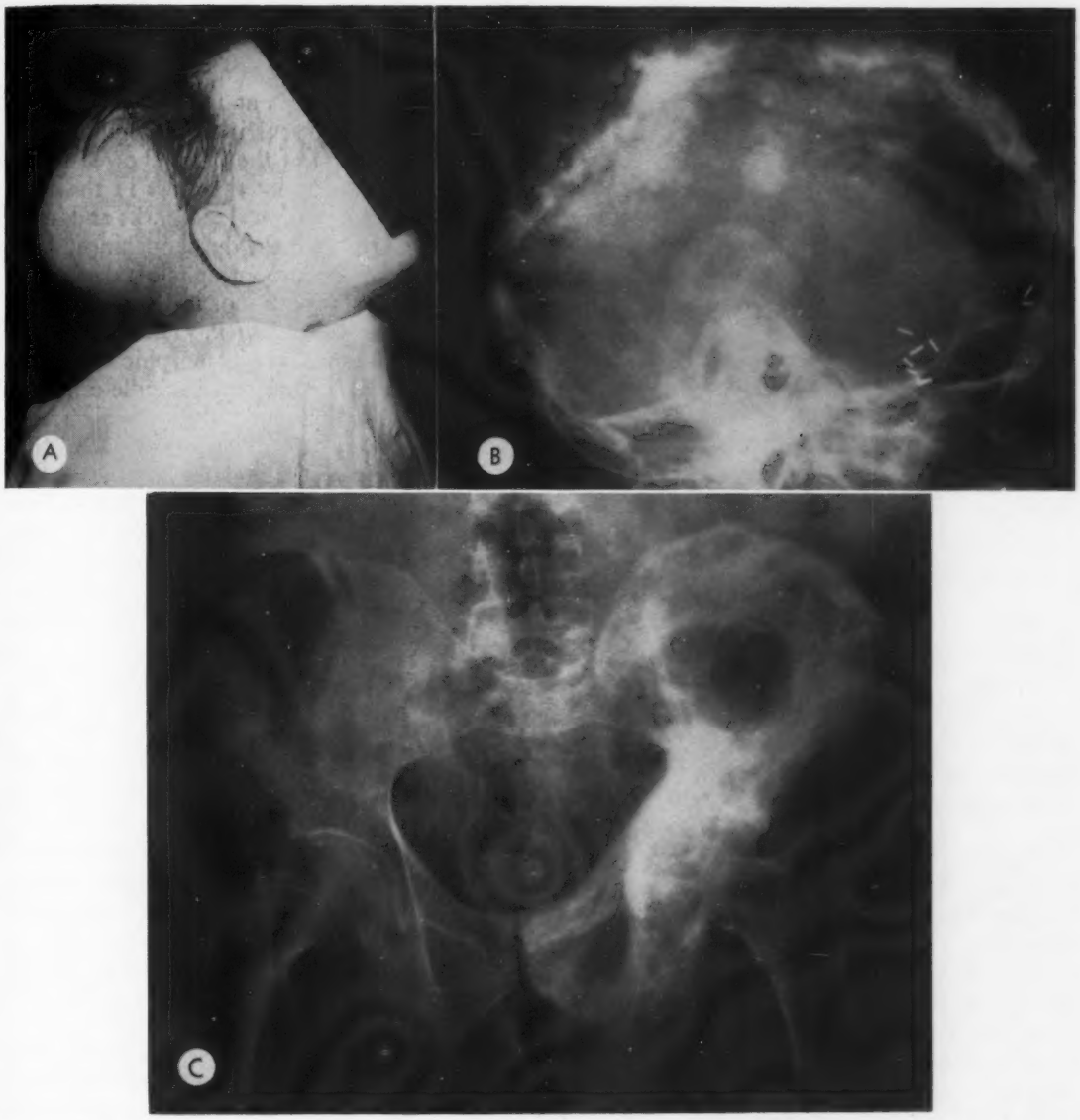

Fig. 1. (*A*) Postmortem photograph of huge occipital mass. Note alopecia of scalp, from previous roentgen therapy. (*B*) Lateral roentgenogram showing Paget's disease of calvarium with concomitant erosion of both tables of skull in occiput. Note soft tissue component of tumor with calcifications. Metallic clips in frontal area represent prior surgery for pituitary tumor. (*C*) Extensive Paget's disease involving left innominate bone.

life of pain, but no regression in the occipital mass was noted. Three weeks prior to current admission the patient developed a right hemiparesis and shuffling gait, followed ten days later by temperature elevation to 100° F. which failed to respond to penicillin. On the day of admission the patient suddenly lapsed into unconsciousness.

Physical Examination. Pulse 110, regular; blood pressure 130:86; temperature 106.4° F. The patient was a well developed, obese, white male in a comatose state. There was a 10 cm. moderately firm, rounded mass protruding from the left occipital region with normal appearing overlying skin, except for alopecia over the area. The pupils were widely dilated and did not respond to light, and there was no corneal reflex. He had diffuse rhonchi throughout both lungs, most marked over the right upper lung anteriorly. All reflexes were markedly de-

pressed, and there was a positive left Babinski sign.

Laboratory Findings. Hemoglobin, 11 gm.; white blood cell count 25,200, with shift to left.

Roentgenographic Findings. The skull at the time of admission revealed typical Paget's disease characterized by zones of increased and decreased density with associated thickening of the tables. The anterior portion of the skull showed "cotton wool" appearance. Metallic clips were noted in the frontal region and the sella turcica was expanded. Of striking interest was a large destructive area involving the occipital region with an accompanying soft tissue mass protruding from the zone of destruction. Bone lysis alternating with osteoblastic components was noted in the occiput. Calcific foci were interspersed throughout the soft tissue component (Fig. 1*B*).

Examination of the pelvis disclosed Paget's disease involving the left innominate bone with marked coarsening of the trabeculae and actual structural enlargement (Fig. 1*C*).

Clinical Course. The patient was started on intravenous fluid containing achromycin in addition to nasopharyngeal aspirations, alcohol sponges, and oxygen tent. Three hours later, without any change in status, he expired quietly.

Significant Autopsy Findings. A 10 cm. apparently encapsulated tumor mass was seen protruding from the left occipital region of the skull (Fig. 1*A*). The mass was completely covered by the galea aponeurotica of the scalp, and the overlying soft tissues were not invaded (Fig. 2*A*). Palpation revealed irregular, soft, almost fluctuant areas, with hard calcific foci within the tumor. When the calvarium was removed, the tumor was found to have eroded the inner table of the skull and to have invaded the underlying cerebral dura (Fig. 2*B*). Tumor protruded from the undersurface of the dura, compressing adjacent brain tissue. Section through the tumor showed dark red, hemorrhagic, friable tissue with minute calcified spicules dispersed in the tumor.

The uninvolved portions of the skull showed marked irregular thickening of the cortical bone, with almost complete absence of the usually evident diploe system, which was replaced by dense grayish-white sclerotic bone (Fig. 2*B*). Similar changes were found in specimens of bone removed from the pelvis.

When the brain was removed from the cranial cavity, a mass was found in a deformed, eroded sella turcica. This at least partly incorporated the right optic nerve and chiasm, flattening these structures and protruded into the lumen of the third ventricle. Gross and microscopic examination of the brain showed the effects of compression by the mass in the skull, with distortion of various landmarks and herniation of gyri through the dural openings. Areas of acute and subacute encephalomalacia were found underlying the tumor in the dura.

Microscopic Examination. The tumor was composed of numerous pleomorphic cells varying greatly in size and shape. Spindle cells were a predominant component although bizarre, irregularly shaped cells were very evident (Fig. 2*E*). Many cells, particularly around foci of osteoid formation, were indistinguishable from osteoblasts. Similarly, benign looking multinucleated giant cells were found in some areas. Tumor cells in mitoses were seen in abundance. The tumor cells were embedded in a loose, reticular, well-vascularized connective tissue stroma. Islands of acidophilic osteoid substance were seen throughout the sections of tumor (Fig. 2*D*). Many of these had undergone, or were undergoing, calcification. Hemorrhagic necrosis of tumor was extensive in the deeper portions of the tumor mass.

Peripheral to the tumor, and in some places within the tumor, were thickened bone trabeculae showing the typical irregular mosaic pattern of cement lines diagnostic of Paget's disease (Fig. 2*C*). Similar findings were present in the samples of bone removed from the pubic rami and left wing of the ilium.

Microscopic examination of the tumor mass from the sella turcica showed a very large chromophobe adenoma, replacing most of the pituitary gland and quite evidently representing a recurrence of the previously diagnosed and treated hypophyseal neoplasm.

DISCUSSION

It is of interest to note the latency of transition in this case. At the time of surgery for a pituitary tumor in 1937, Paget's disease of the skull was present. The eighteen year period is not unusual in observation of malignant change in this disease. For this reason, periodic follow-up examinations for possible signs of malignant transformation are indicated in any patient

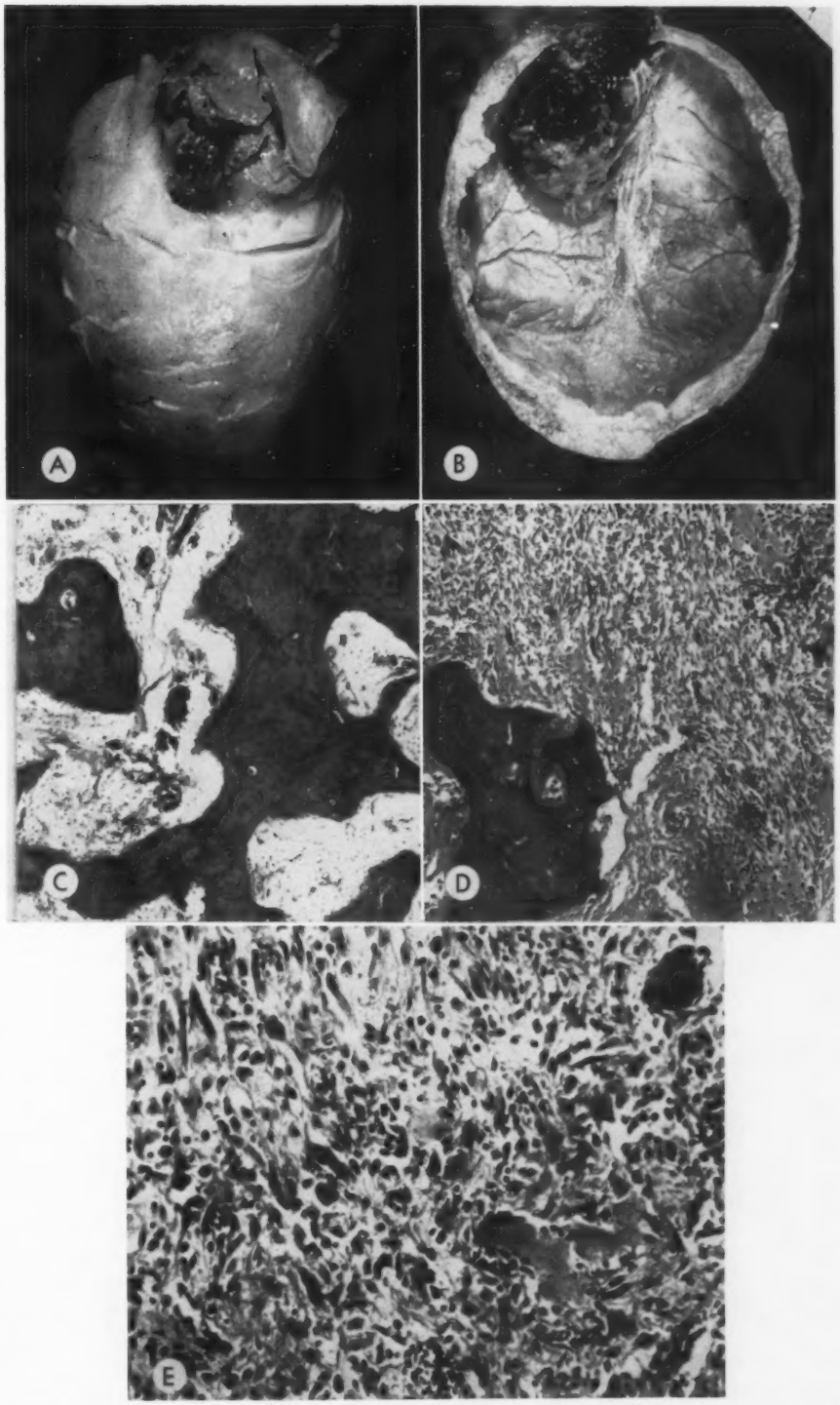

FIG. 2. (*A*) Outer surface of calvarium with protruding tumor mass. (*B*) Inner surface of calvarium with destruction of inner and outer tables and extension of tumor mass. Note markedly thickened bones of calvarium typical of Paget's disease. (*C*) Photomicrograph of Paget's disease in skull. Note irregularly thickened bone trabeculae and mosaic pattern of ossification (×85). (*D*) Photomicrograph of skull showing Paget's disease (lower left) and osteogenis sarcoma (×85). (*E*) Photomicrograph of tumor showing bizarre cells and foci of osteoid formation (×300).

with Paget's disease, regardless of how slight the involvement may be. Sherman and Soong[7] have called attention to the difficulty in diagnosing malignant transformation of Paget's disease in the skull. The diploe is destroyed and there is often soft tissue extension. Both osteoblastic and osteolytic processes are seen simultaneously. Tangential roentgenograms have been suggested to evaluate destruction of the tables due to soft tissue extension. In the above case, the skull was not the sole area of involvement with Paget's disease. However, malignant transformation in a solitary site has been observed.

SUMMARY

1. An unusual case of Paget's disease of the skull with malignant transformation into osteogenic sarcoma has been described.

2. The latency in the appearance of this transformation and the difficulty of making an early diagnosis have been emphasized.

3. Of incidental interest is the coexistence of a recurrent pituitary adenoma.

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PRIMARY RETICULUM CELL SARCOMA OF THE SKULL*

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RETICULUM cell sarcoma occurring primarily in the skull is a rare and unusual entity. A review of the literature disclosed only 2, possibly 3, similar cases, reported by Strange and de Lorimier in 1954.[6] Their first 2 patients are alive and well over nine and three years after treatment, respectively. However, the situation reported in their Case III differed from that in the other 2, and as the patient died some two and one-half years after the onset of the condition de Lorimier[2] now thinks it likely that the cranial lesion may have been metastatic rather than primary.

The patient whose illness is recorded here has been under observation for five years. There seems little doubt that her multiple cranial lesions were primary ones. They responded most satisfactorily to excision and roentgen therapy. There has never been any evidence of similar tumors elsewhere. Her history of serious pulmonary tuberculosis made clinical differential diagnosis difficult.

REPORT OF A CASE

G.B. was a fifty-three year old white female who came to The Chicago Memorial Hospital in August, 1952 complaining of pains in her head of eight months' duration.

She had suffered from severe and extensive pulmonary tuberculosis which had been treated with bilateral pneumothorax. In 1947 a left thoracoplasty was made. Subsequently repeat examinations of the sputum for tuberculosis have been negative and a roentgenogram of the chest has revealed no change since 1947.

In December, 1951, she experienced sudden, sharp, recurrent, left occipital pains which could be precipitated by cold drafts. After two weeks similar pains occured in the left frontal area associated with some swelling of the soft tissues. She remained afebrile and without systemic complaints, obtaining some relief with mild analgesics. When the swelling developed in the left frontal region the patient was placed on antibiotics and in a short time the swelling is said to have subsided. The left frontal and occipital pains persisted unaltered, still being precipitated by cold drafts or cool water, so that she avoided shampooing her hair.

The positive physical findings with the exception of the residuals of the thoracic surgery were confined to the skull. There were areas tender to light palpation 8×9 cm. in size in the left frontal, and 5×5 cm. in the left occipital portions of the skull. The scalp was slightly thickened only over the anteromedial portion of the tender zone in the left frontal region and in an area about 2 cm. square. There was no other tumefaction, local elevation of skin temperature, sensory deficit, trophic or vasomotor changes of the scalp. Neurologic examination was entirely negative.

Routine hemogram, urinalysis, and serologic tests were negative. Examinations of the sputum were negative for tubercle bacilli.

Serial roentgenograms of the skull made in March, July, and August of 1952 revealed a progressive bone destruction in the left frontal and left occipital bones (Fig. 1 and 2). This was interpreted as compatible with a low grade osteomyelitis. The roentgenogram of the chest revealed only the changes in the upper ribs on the left side resulting from the thoracoplasty. There was a collapse of the upper portion of the left lung. The remaining lung fields were clear except for areas of calcification on the right side.

Because of the history of severe pulmonary tuberculosis and of the presence of soft tender areas in the left occipital and left frontal regions, with roentgenologic evidence of irregular destruction of the bone in those areas, a diagnosis of tuberculous osteomyelitis of the skull was made and radical extirpation of the involved bone was advised. On August 6, 1952 a left frontal craniectomy revealed a mass of granulation tissue measuring 2×3 cm. attached to the periosteum which was excised. Softened and eroded bone was rongeured away until

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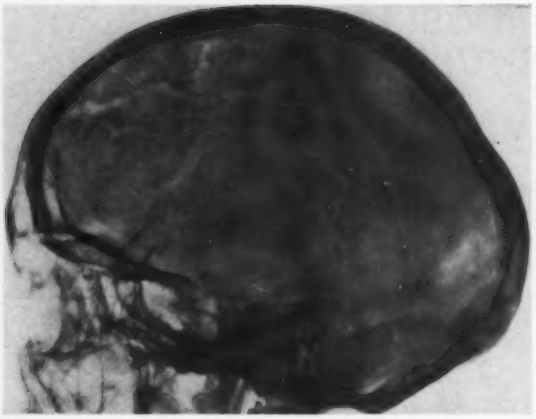

FIG. 1. Roentgenogram of the skull made July 16, 1952, revealing patchy decalcification of the skull in the left frontal region just anterior to the coronal suture and in the left occipital region just above the inion.

normal appearing margins presented, leaving a defect measuring 8×9 cm. (Fig. 3). The dura mater did not appear involved in this pathologic process. The left occipital lesion was treated in the same manner on August 11, 1952. The defect made in the skull measured 5.5×6 cm. (Fig. 3). It extended 3×2 cm. to the right of the midline. Some slight calcification was noted in the underlying dura. The pathologic specimens were brown, soft, friable, and occasionally contained bony spicules. Bacteriologic examination of these tissues, including special studies for tuberculosis, were negative.

FIG. 2. Roentgenogram made less than a month later on August 5, 1952, demonstrates a striking progression of the disease.

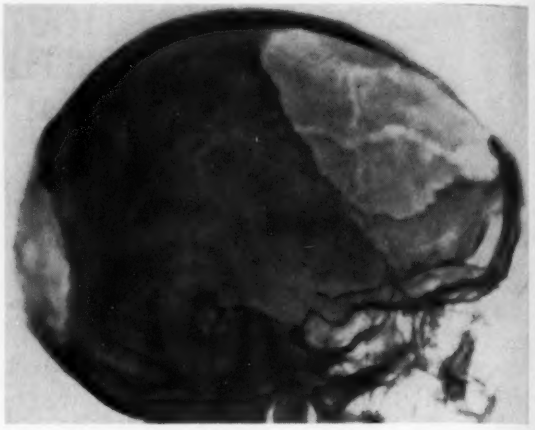

FIG. 3. Roentgenogram of the skull made on August 8, 1952, shortly following the operation demonstrates the surgical defect.

The microscopic examination, as reported by Dr. Otto Saphir and Dr. Eleanor Humphreys, disclosed a tumor composed of closely packed pleomorphic cells typical of a reticulum cell sarcoma (Fig. 4). The cells were imperfectly subdivided by trabeculae and small blood vessels which were accompanied by a scanty collagenous stroma. This loose stroma was reinforced by the characteristic network of fine

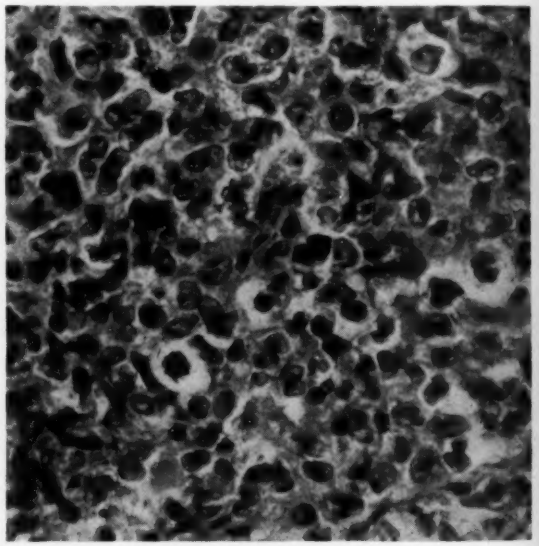

FIG. 4. Photomicrograph of the tumor showing the densely packed pleomorphic cells with numerous mitotic figures. (H. and E.×450.)

reticulin fibers which were well demonstrated by impregnation with silver (Fig. 5). The cells of the tumor ranged from ordinary macrophage-like and fibrocyte-like cells to large cells with lobated or multiple nuclei. In some the nucleoli were quite large. Mitotic figures were numerous. They were found in all stages. Many of them were bizarre. The bone marrow had disappeared from the region of the tumor. The few small dark cells present were lymphocytes and plasmocytes. Bone persisted focally, especially near the margins, as eroded trabeculae or necrotic fragments (Fig. 6). Occasionally bits of reactive new bone were seen. The histopathologic diagnosis was typical, highly malignant reticulum cell sarcoma.

Postoperatively the wounds healed without incident and except for minimal discomfort over the incisional sites there were no complaints.

A roentgenogram of the skull one month postoperatively showed no changes from the roentgenograms made immediately after the operation, but two weeks later there were minimal marginal changes of erosion in both craniectomy sites similar to the original lesion. A general roentgenologic survey of the skeleton was made at this time but no other areas of neoplastic involvement were found. On September 28, 1952, roentgen therapy was begun and given over a two-week period in divided

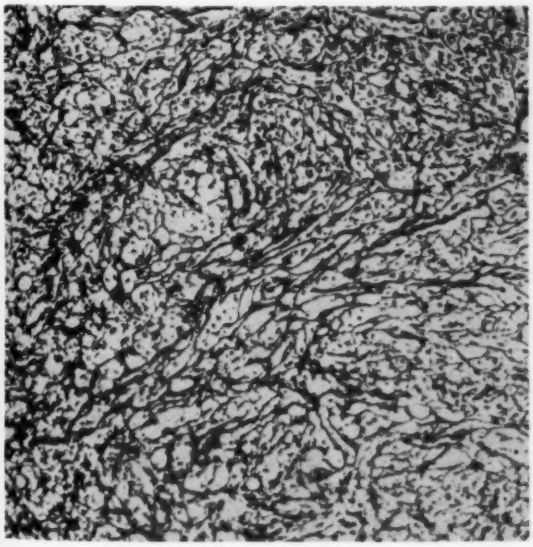

Fig. 5. Photomicrograph showing the intercellular reticulin network. Silver impregnation. (×115.)

Fig. 6. Photomicrograph showing the spicules of bone among the densely packed cells of the tumor. (H. and E. ×122.)

Fig. 7. Roentgenogram made on May 9, 1956, showing a limited amount of regeneration of bone along the medial and inferior margins of the bony defect.

doses to the craniectomy sites. The estimated dose to each locus was 3,500 to 4,000 r (0.9 mm. Cu half-value layer). At three-month intervals repeat roentgenograms of the skull showed progressive but limited regeneration of bone in the defects.

In June, 1955 roentgenograms of the skull and chest were unchanged. A general roentgenographic examination of the remainder of the skeleton was again normal. Studies of the bone marrow, spinal fluid, and blood proteins were all within normal limits as were the routine studies of the blood and urine. At this time her only complaints were occasional mild headaches, which were relieved by aspirin.

The last re-evaluation was on May 9, 1956 at which time the roentgenograms of the skull continued to remain unchanged (Fig. 7). Repair of the defects in the skull has been discussed with the patient but in view of her continued good health she has elected to leave these defects as they are.

In a letter dated February 17, 1958 the patient stated that in October, 1957 she had suffered "a slight stroke on my right side." Subsequently this condition improved so that she did not think it necessary to return before her annual check-up in May, 1978.

Comment. The fact that this woman has gone over five years in good health without recurrence of her tumor and without its having developed elsewhere would seem to leave little doubt but that these were primary, multicentric lesions and that the tumor has been eradicated. The prognosis, particularly in the light of the cases reported by Strange and de Lorimier,[6] would appear to be excellent.

DISCUSSION

Previously described roentgenologic findings in the skull with reticulum cell sarcoma include diffuse mottling resulting from deossification, dissolution of the outer table, sequestration and evidence of erosion of the outer aspect of the inner table. In general, this description is no different from the roentgenologic appearance of reticulum cell sarcoma occurring in other portions of the skeleton. Strange and de Lorimier[6] in recording their 3 cases delineated the above. A personal communication was received on June 30, 1955 regarding their cases. The patients of Cases I and II were treated with roentgen rays in April, 1946 (1,000 r minimal tumor dose) and February, 1952 (1,800 r minimal tumor dose), respectively. Both of these were "in excellent condition." The patient in Case III had died after a period of about two and a half years, suggesting the possibility that this was a metastatic rather than a primary lesion. Excluding Case III there are some similarities and dissimilarities with our case. All were females, afebrile and nontoxic. There were no abnormal laboratory findings other than the roentgenograms of the skull. Strange and de Lorimier[6] report single, painless, nontender soft tissue swellings while in our case the patient complained of painful tender areas and the lesions were multiple. In addition, tumefaction was evident in only a portion of one of two separate tender areas. It should be noted that swelling of the scalp had occurred in this case and whether this developed because of thrombosis or phlebitis of the interdiploic veins is speculative. The roentgenologic appearance, although fairly typical, cannot be readily differentiated from that of a chronic inflammatory lesion of the calvarium. A positive diagnosis can be made only by microscopic examination.

As to the treatment of reticulum cell sarcoma in other bones of the skeletal system there is no unanimity of opinion and experience here is of little value as a guide in the treatment of similar tumors limited to the skull. Roentgen therapy (2,400 to 4,000 r tumor dose), excision or amputation or the administration of Coley's toxin have been employed either separately or in combination and with varying results in the treatment of reticulum cell sarcoma in bones other than the skull.[3,5]

The treatment of choice in reticulum cell sarcoma of the skull would appear to be radiation therapy once a positive diagnosis is established, to judge from the experience of Strange and de Lorimier.[6] Furthermore, the dose required does not appear to be great (1,000 r and 1,800 r in their cases).

Their experience indicates that there is no need for a radical excision of the lesion such as was made in our case. However, this small series of cases is obviously inadequate to permit a final decision on this matter. In any event, it would appear essential to establish the diagnosis by a biopsy of the skull before irradiation is undertaken.

SUMMARY

A case of multiple primary reticulum cell sarcoma of the skull is described. Following excision and roentgen therapy she is now well for over five years. Her history of severe pulmonary tuberculosis made clinical differential diagnosis difficult.

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THE ROENTGENOLOGIC CRITERIA FOR APPRAISING THE HUMAN BACK AS AN ECONOMIC ASSET OR LIABILITY*

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BACKACHE is responsible for a considerable part of medical disability in any community. The vast majority of industrial backache in America concerns the low back. Pre-employment roentgenographic studies of the lumbosacral spine have been instituted in an attempt to reduce its economic significance. In the period of ten years since the first report of this procedure was made by one of the authors,[13] this type of examination has been successfully applied over a wide area.[1,2,3,10,14,18] Little attention, however, has been given to the criteria for evaluating the individual applicant. It is the purpose of this paper to dwell in detail on the criteria used in our practice. A subsequent communication will be devoted to the evaluation and results of our pre-employment studies.

Economically, a back may be an asset or liability to both the owner and employer. It would seem reasonable, therefore, to attempt to appraise a back's potential economic value at the earliest possible moment. From the owner's standpoint it should be done before he makes the fateful decisions of adolescence—quitting school, majoring in physical education, etc. The employer can only protect himself and the applicant when the latter appears at the employment office. It is obvious that the examination at fifteen years of age will not reveal the back at the age of forty-five, and the most perfect back may well be totally wrecked by a major accident, or temporarily incapacitated by some minor movement.

The whole problem requires understanding by employer, applicant, and physician with recognition of the limits placed on each.

The *employer* should recognize that even the most highly qualified medical judge working with the most technically perfect tools cannot foresee the future, the accident, the rate of aging, the onset or the nature of disease; he can only render judgment on the evidence presented.

The *applicant* must realize that race, religion, political affiliations, and associations are too superficial to be detected by roentgenograms, perhaps even by examination. He should likewise be aware that the employer is legally permitted to protect himself from liability by requiring any reasonable examination of the applicant that the employer deems necessary to secure that protection.

The *physician* must realize that the job applicant generally assumes his own physical perfection and that his rejection is likely to cause a resentment that should be resolved by an explanation. This must be in lay terms, taken from his own field of reference. Medical terms are not understood and may be frightening. The bigger the terminology, the worse the disease.

The physician, whatever his specialty, who determines the employability of an individual, is sitting in a position as fateful, so far as the applicant is concerned, as that of the Chief Justice of our Supreme Court. The judgment of both must be unswerved by irrelevant considerations, and the medical judge, like his legal counterpart, should permit only strict medical evidence to be brought into his judicial chambers. The criteria should be demonstrable, rational and generally acceptable to men skilled in making these judgments. The recorded judgment, while it may detail a

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great mass of information in medical terms, should be summarized in a clear lay word or two, *e.g.*, acceptable or unacceptable. We prefer good, fair and poor risk, which puts both employer and applicant on a graded system of "alerts." Backs have been graded by the writers as:

Good risk—no demonstrable defect—employable for any job requiring physical strain or effort.

Fair risk—defects present, but of such a nature as to permit his employment for labor, but not for repetitious or continued heavy low back work. (Questionable fair—this rating was used only during World War II when labor was at a great premium and has since been discarded.)

Poor risk—defects present of such magnitude as to render inadvisable lifting or heavy labor.

Employer, applicant and physician should all recognize that the back fit for stevedoring is not required for watch repairing, but a back might be so bad as to render an individual unacceptable for the lightest work—tuberculosis, malignancy, etc. Usually individuals so affected are seeking medical aid rather than employment. Obviously the examiner should be acquainted with the job requirements. It is to be reiterated that the criteria of risk classifications in each individual are applicable only if the labor involved concerns heavy low back work or stress. The probability of serious breakdown in the poor risk group is then sizable and significant. On the other hand, the application of these criteria to other working individuals, in occupations not having such low back stress, is not valid.

An employer who requests an applicant to lift a sack of grain or cement reveals the applicant's skill and his ability to lift a specific object. The strength of the mechanism could be graded by a weight-lift machine, comparing the findings with age, height, weight and experience tables. Lifting a heavy object would also permit the employer to appraise the individual's method of lifting and to correct the same before employment. Such a procedure would not reveal the hidden flaws in the human machine that potentially may produce a breakdown in the future.

As the flaws in a casting are revealed by roentgenography and other physical methods, so the only satisfactory method of making an appraisal of the hidden parts of the spine is by roentgenographic examination. This has been done by us on 6,010 individuals during the past fourteen years, using a concurrent series of patients complaining of backache for statistical comparison.

The techniques found satisfactory, the standards of judgment, and the method of rating will be described. It is proposed to: 1. Outline the roentgen examination of the low back that was found satisfactory; 2. describe each anatomically functional unit indicating (a) its normal function, (b) pathologic anatomy revealed by roentgenograms and the consequent changes in function, (c) our estimate of the degree of disqualification for each condition. 3. A statistical analysis of the patients that have been examined will be the subject of a future communication.

THE ROENTGEN EXAMINATION

Ordinarily five views are required for such an appraisal, but sometimes twice this number may be necessary. However, if the roentgenologist is able to critically examine the wet films, it is possible in 60 per cent of examinations to be satisfied with three views: (1) an anteroposterior view of the lumbar spine and sacrum that is centered just above the iliac crests; (2) an anteroposterior view with the back flattened on the table by hip-knee flexion, the knees spread and the central beam angled 20 degrees toward the head, and centered on the lumbosacral junction; (3) an erect lateral roentgenogram centered over the lumbosacral junction. It must include at least the third lumbar vertebra and the upper half of the sacrum. Fixation bands are mandatory for roentgenography in the lateral view. Additional films are secured if there is the least question of doubt.

The Bucky diaphragm is employed routinely and every view is "cone cut" at all

corners. The smallest anode focal spot consistent with tube safety is used with maximum milliamperage to give the shortest possible exposure time. We insist on small views to avoid the obscuring of fine detail by scattered radiation, which reaches its maximum in the heavily muscled individual. Motion study roentgenograms with the individual bending laterally have not been used, but flexion and extension lateral roentgenograms have been most helpful in certain problem cases.

ANATOMICALLY FUNCTIONAL UNITS

I. THE BASE

The base upon which the semiflexible spine is erected is the pelvic ring, normally a symmetrically distributed structure curving outward and forward from the spine. It recurves to mutually brace itself at the symphysis pubis. This base may be extended upward through the spine to any desired level by the postural activity of the appropriate muscles. Sacral spina bifida may be considered a basal defect, but otherwise a defective pelvic ring or base has been found so rarely that its effect on employability will not be discussed.

II. THE FULCRA

The facets of the pars intermedia are a curious combination of fulcra and levers, except the sacral pair which are purely fulcra. Owing to these mixed functions, the fulcra are not fixed as in the ordinary mechanism but are slightly movable on each other and are strangely compound. Because of this mobility the fixation at any particular level must be done by the postural activity of the muscles below that level. The descending facets of the vertebra above hook behind the ascending facets of the vertebra below like the flukes of an anchor and permit the supraincumbent spine to swing safely at anchor. At each level motion normally may occur in one or more planes. For security each set of articulating facets must lie in the same plane, parallel planes, or be arcs of the same circle.

A. The most insignificant fulcral defect is a lack of development or failure of fusion of the tip of an articular facet. This may be so slight as to be of no economic significance. On the other hand, we sometimes find a facet so rudimentary or absent that we judge a man disqualified from heavy labor (Fig. 1).

B. Asymmetric facet. Goldthwaite[8] nearly a half century ago pointed out that a pair of facets not infrequently are dissimilar —one planar, the other crescentic; one facing sagittally, the other frontally; one planar, the other conical, or eccentrically crescentic or conical. When this asymmetry exists, wear and tear on the joints, ligaments and disks of this level are abnormal and have manifested themselves in our examinations by zygapophysial degenerative changes, thin disks and vertebral body spurs. Clinically, the association of asymmetry of the facets and backache is extremely common in our experience. The nearer the pelvic base, the more the strain and the greater the clinical significance. Asymmetry of the facets in the upper lumbar spine alone has a prognostic significance that is too elusive to be assessed. Minimal asymmetry at the L_{4-5} level or lumbosacral junction is apparently in the same category. Appreciable or 90 degree asymmetry at either of the latter two levels is deemed a fair risk. If accompanied by even minor vertebral body spurs, zygapophysial facet sclerosis, or other degenerative phenomena, the risk is considered poor.

C. Spondylolysis and spondylolisthesis. This most serious defect of the spine usually occurs in the fulcra of the last three lumbar vertebrae. The lesion or defect is observed between the superior and inferior facets of one or more vertebrae in the pars intermedia on one or both sides. Spondylolysis or a "solution of continuity" differs connotatively from spondyloschisis—a splitting of the spine. Lysis connotes an insidious process of separation or nonunion; schisis a dramatic or traumatic fissuring of continuity; while listhesis denotes a slippage. In not one of our backache or preemployment cases of spondylolysis or spon-

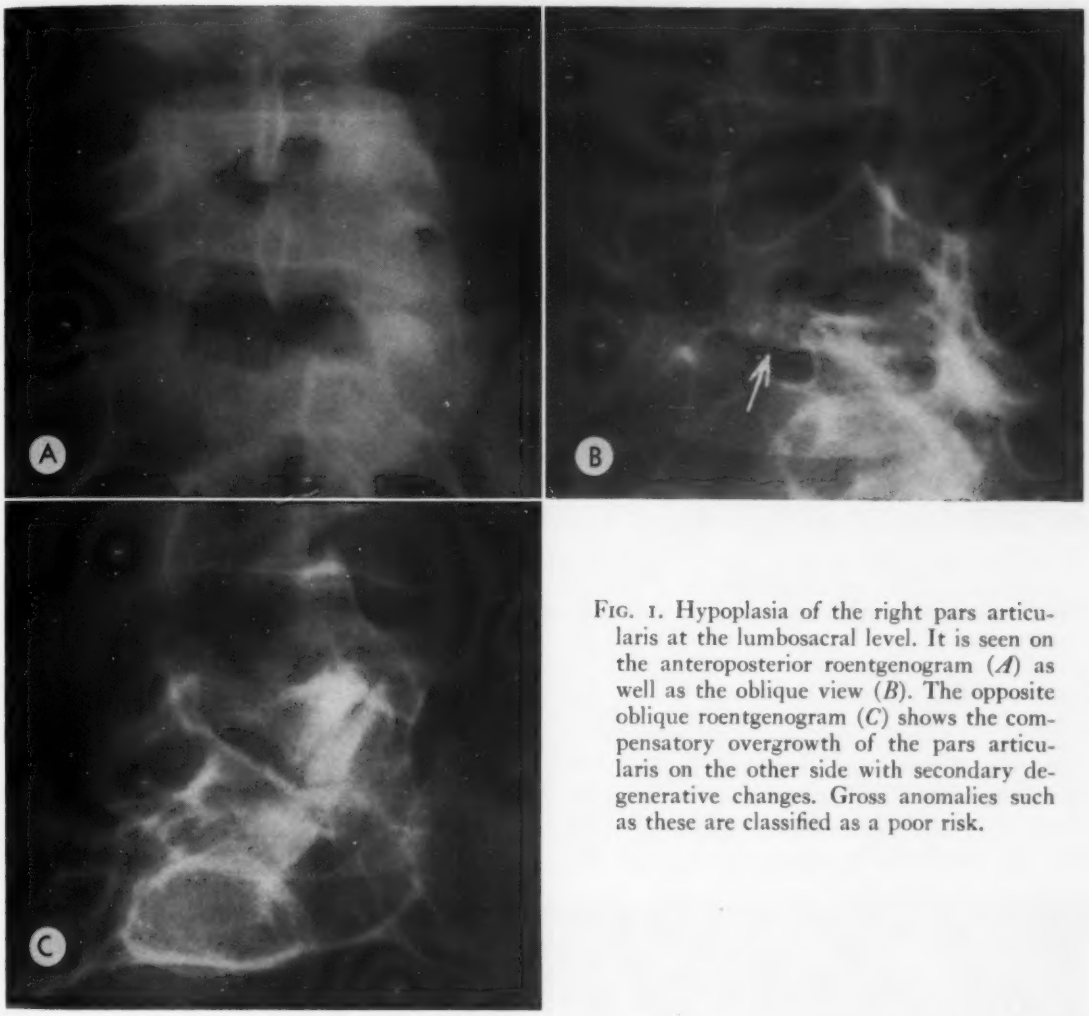


Fig. 1. Hypoplasia of the right pars articularis at the lumbosacral level. It is seen on the anteroposterior roentgenogram (*A*) as well as the oblique view (*B*). The opposite oblique roentgenogram (*C*) shows the compensatory overgrowth of the pars articularis on the other side with secondary degenerative changes. Gross anomalies such as these are classified as a poor risk.

dylolisthesis was a previous history of significant trauma or an initial period of incapacity obtained. Therefore, we prefer the term of spondylolysis, although perhaps better would be some negative term that connoted an embryonic or developmental failure of union such as spondylodysgenesis, rather than one that points toward an active process, even as mild as dissolving.

Spondylolisthesis or spondylodysgenesis can usually be seen on one or all of the routine roentgenograms. If it is suspected or if the laminar areas are unsatisfactorily presented, 45 degree oblique roentgenograms are obligatory. Occasionally, oblique views with the central ray angled 20 to 30 degrees in a cephalic direction are needed to unfold this area. The flexion lateral roentgenogram may be helpful. The usefulness of this view was discovered when a case was found to have a bilateral L_5 spondylolysis at surgery, although the films including 45 degree oblique roentgenograms failed to show the defect, even in retrospect. On reviewing the roentgenograms, the defect was humiliatingly obvious only on a lateral flexion diskogram (Fig. 2). Since that time a lateral erect flexion roentgenogram has been made frequently and it often demonstrates the defects with clarity (Fig. 3). This is particularly true when motion or spreading occurs between the detached portions. Associated with this defect other abnormalities are frequently seen, such as

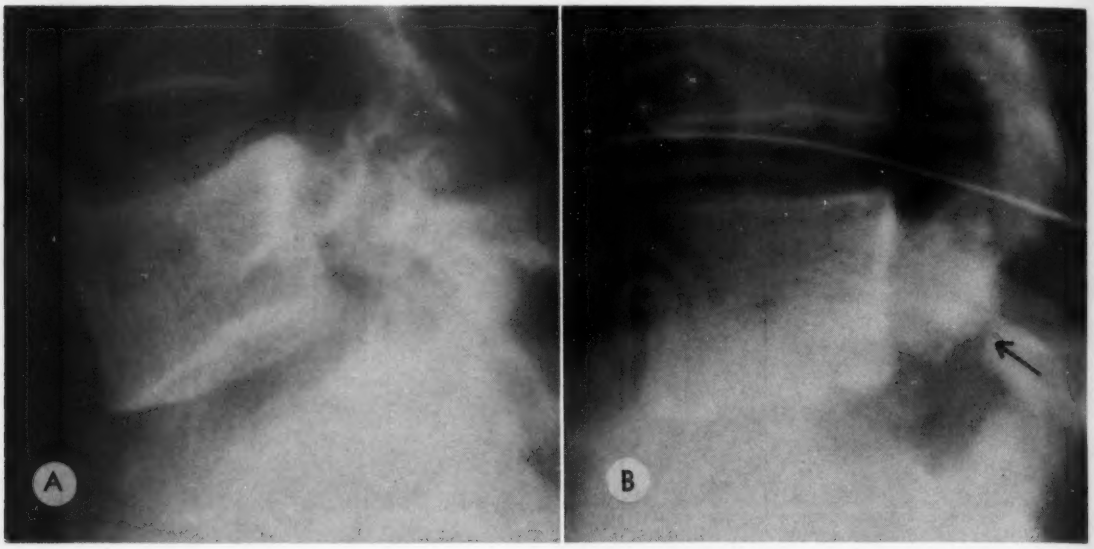


FIG. 2. The spondylodysgenic (lytic) defects of the pars media are not clearly seen on the erect lateral roentgenogram (*A*), but are clearly seen on a lateral flexion roentgenogram done during diskography (*B*). In this case the bilateral defects were not demonstrable on the 45 degree oblique views, where one expects to see them.

narrowing of the intervertebral space, degenerative changes in the vertebral bodies and zygapophysial articulations; and the laminar area, particularly in the sacrum, may be largely defective or the area near the defect may be sclerotic. This latter finding has been considered by some to be callus. To the writers such an interpretation appears fictitious, since in most instances the younger individuals rarely present such a change, and the sclerosis requires years to appear, if it develops at

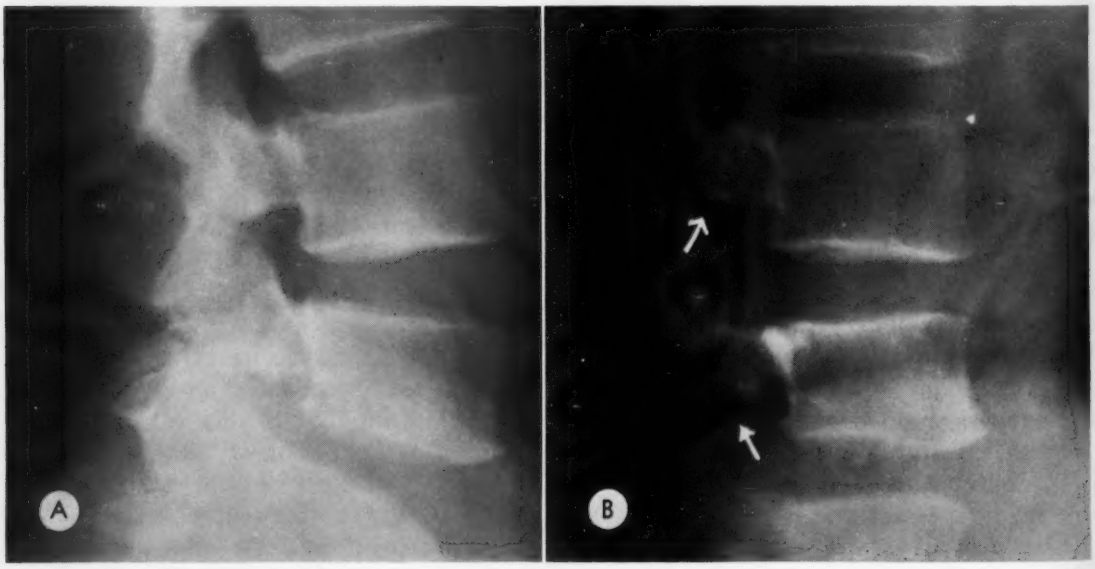


FIG. 3. Double spondylodysgenic (lytic) defects of the pars media of the lamina at both the L_4 and L_5 levels, which were visible at the lower level on the lateral roentgenogram (*A*), but ideally shown by the flexion lateral roentgenogram (*B*) at the L_4 level. On the flexion lateral roentgenogram the laminar defects may be seen with clarity when not demonstrable on the other views.

all. The high incidence of associated lytic defects in the contiguous spinous processes is also suggestive of a dysgenesis. Other conclusive points disproving fractures as an explanation are adequately covered in the literature.[6,16] The demonstration of a spondylolisthesis or a spondylodysgenesis (unilateral, bilateral or multiple) places the individual categorically in a poor risk group.

III. THE LEVERS

Each of the 24–26 individual levers is composed of the following elements:

A. A weight-bearing arm lying anterior to the fulcra or articular facets, consisting of the vertebral body, the intervertebral disk, and the short pedicles that transmit the weight-bearing strain to the fulcra.

B. The motor or force arms made up of several parts which may be effective at any moment or not, depending on the particular act required.

1. Posteriorly there are the Y-shaped laminae and spinous processes.
2. Laterally in the cervical and lumbar areas there are the transverse processes.
3. In the dorsal region the transverse processes and rib cage provide lateral and anterolateral levers.

A. The *vertebral body* is the weight-bearing portion and structurally it must be adequate for the task and yet conform to the nature of a living substance. As we pass downward, the body weight increases slowly and the stresses and strains increase correspondingly; therefore, there should be and is a gradual increase in the size of the elements. The internal architecture and density of the individual vertebrae must be adequate to carry the load.

One of the commonest pathologic changes observed is a difference in vertebral height, usually at one margin of the projection more than another, producing a wedge which disturbs the whole supraincumbent mechanism. This tends to increase the postural muscular power required, that results in earlier fatigue, decreased efficiency and even pain. The most common causes of this loss of height are fractures, followed by destructive disease, and the congenital anomalies. All are well recognized by the radiologist.

Wedged old healed fractures of the vertebral bodies in the lumbar and lumbosacral spine require individual evaluation. A general approach is as follows: Complete healing of an old fracture of the upper lumbar spine with only mild wedging is considered of minimal significance, although a change of this nature at the fourth and fifth lumbar level would place the individual in the poor risk category. Wedging and ligamentous calcifications in the upper lumbar spine with an associated kyphosis or scoliosis would place the case in the poor risk group. Variations between these two types would be judged accordingly.

There are several conditions affecting the vertebral bodies that are not commonly recognized for what they are: (1) Amphicelous vertebrae. Any who examine what they eat with their eyes as well as their nose and tongue will have observed that in fish the opposing vertebral surfaces are concave. Every once in a while that condition is observed in man with vertebrae of normal density. In our experience and judgment this in no way impairs a man's usefulness to himself or his employer, provided the osseous density is normal (Fig. 4). (2) Limbus bodies. The limbus body is frequently mistaken for an old ununited fracture in that it resembles the wedge-shaped piece pushed off the vertebrae in milder fractures (Fig. 5). It resembles the intercentra of primitive amphibians such as Seymouria. Careful roentgenographic examination will show a smooth border all around the triangular cross-section of the body. Viewed from above, the body would be crescentic. This likewise in our judgment is of no significance. (Three separate cases with backache have been seen in the last year with symptoms localized over a vertebral segment having a limbus body. This has aroused our suspicions of its potential

Fig. 4. Amphicelous concave vertebral plates, particularly marked at the lumbosacral level. This finding has no clinical nor prognostic significance.

importance and further thought will be given to this in the future.) (3) The following asymmetrically shaped vertebral bodies are also considered to be without significance: those with a hatchet or reverse wedge shape (the anterior height is greater than the posterior height), caudally foreshortened bodies, and those that are smaller than the segment above. (4) The slope of the upper border of the sacrum, the lumbosacral angle, is variable. An increase in this angle beyond 40 degrees with the resultant exaggerated lumbar lordosis has been considered abnormal by some authors.[5] Our experience has not borne this out, and hence we have felt obligated to presume it a normal variant.

Old Epiphysitis of the Scheuermann's type. Evidence of this entity in the lumbar spine is not uncommon. When the findings are confined to the upper lumbar area, with only moderate residual irregularity to the anterior portions of the vertebrae and Schmorl's nodule formation, the candidate is usually passed as fair. We believe similar findings at the lumbosacral level create an increased risk. If the disk spaces are narrowed or the irregularity is at all appreciable, we classify the candidate as a poor risk. We have not seen enough individuals in this category to give a firm statistical prognosis as to long term follow-up, but the above seems valid.

The general density of the vertebrae is of significance; osteopetrosis with its chalky white bones and osteoporosis with its gray washed out appearance. Neither seems to have the inherent strength that heavy labor requires. Fortunately neither shows up with frequency in the pre-employment examinations. Segmental increased or decreased density is a danger sign, as exemplified by hemangioma, osteitis deformans, cystic lesions and neoplasms (Fig. 6). These all call for a rating of poor risk.

A common architectural disturbance is the presence of cartilaginous intrusions into the vertebrae, the so-called Schmorl's nodes. The significance of these will be discussed under the intervertebral disk.

The number of transitional lumbar vertebrae is not considered significant if the process is complete. It appears to make no measurable difference whether there are 4, 5 or 6 lumbar segments.

Fig. 5. Classic limbus body, which is without significance and by itself is considered a good risk.

The second portion of the weight-bearing mechanism of the spinal levers, the *intervertebral disks*, are essentially flexibly jacketed, semifluid vertebrae the shape of which changes at every move, thereby permitting motion. Anything that affects this adaptability impairs their usefulness. These changes may affect the flexibility of the jacket or the moldability of the nucleus. The common changes in the jacket are minute tears which are replaced by calcareous or bony deposits that shorten or limit motion at that particular level. These can now be seen in roentgenograms as spurs or ligamentous calcifications. This reduction in mobility increases the likelihood of further damage under even smaller strains and the condition may be progressive. It is probable that some of the basic change affects the elasticity or fluidity of the nucleus so that it becomes stringy and fibrous.[11]

A tear or rupture of the first may permit an extrusion of the latter. Any such tear or extrusion is not immediately apparent roentgenographically, but becomes manifest after a time as a decrease in the height of the intervertebral space and the neural foramina, and abnormal overlapping of the articular facet.

An extruded nucleus is gradually absorbed and the person may be clinically symptom-free, but a narrowed intervertebral space, particularly in a young person, and especially at the L4–5 and L5–S1 levels is a warning that the prospective employee is unfit for heavy labor, and they are classified as poor risks, be it from degeneration or herniation.

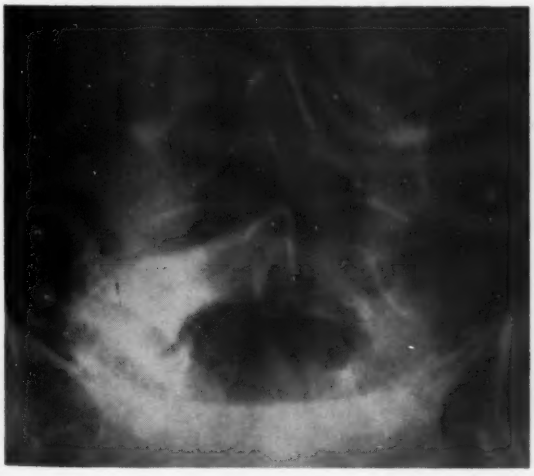


Fig. 6. Proved fibrous dysplasia of the lamina and pedicle. Such changes in pre-employment evaluations are considered a poor risk even though the exact microscopic diagnosis in other cases may not be certain.

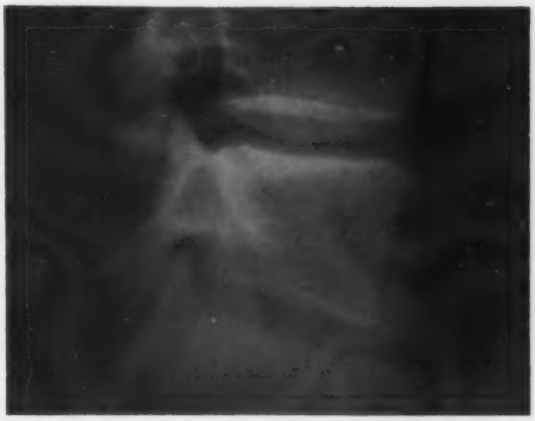


Fig. 7. Obvious gross thinning of the L4–5 intervertebral space with associated degenerative phenomena.

Narrowing of an intervertebral space above the L4–5 level is not considered of such great prognostic importance unless small vertebral body spurs are also present; then the risk is considered fair.

When is an intervertebral space narrowed? It is our conviction that the space between L4 and L5 in the normal young adult is the widest space in the lumbar region and should always be equal to or wider than the space above (Fig. 7 and 8). This "yardstick" is used to evaluate the width of this space. Numerous authorities[17] have disclaimed the wisdom of this "measuring stick," primarily because narrowing is then present in a large portion of the older working male population. It is our feeling that these individuals have degenerated disks, and such surveys have only pointed out the frequency of this finding in older laborers, bearing out Lindblom's[9] conclusions. A degenerated disk in an older working male obviously is a fairly common finding, and it is not necessarily associated with a disabling low back, or even current symptoms

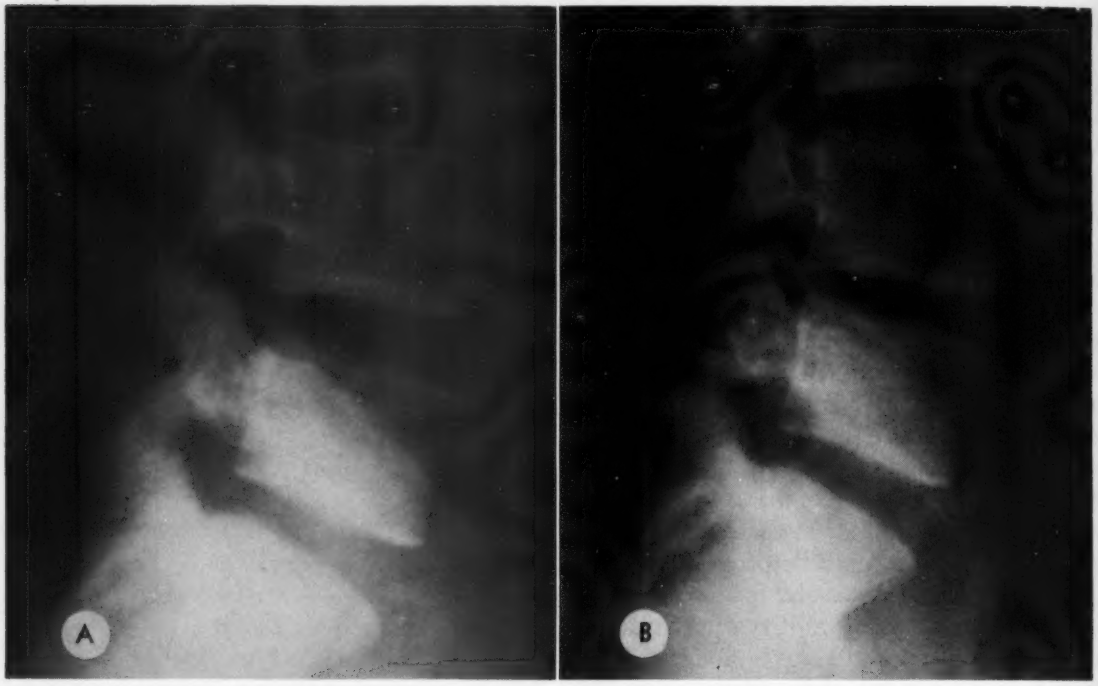


Fig. 8. (*A*) Normal L_{4-5} intervertebral space which is narrowed five months later. (*B*) Although this example is from an individual complaining of backache, it ideally demonstrates that in the normal individual the L_{4-5} space is wider than the space above. It also points out that time is required before this narrowing occurs, as this individual had back and leg pain with an onset just before the exposure of (*A*) with the same symptoms persisting at the second study five months later.

at all. In the younger individual a degenerated disk we feel carries a more serious connotation, predisposing him to herniation of the nucleus pulposus. In our cases, it is remarkable how often a backache history is available, albeit often only after the individual knew of his poor risk classification. Perhaps of more significance is the satisfactory use of this "measuring stick" in the backache group of patients where the incidence is 4 to 1 as compared to the presumably normal pre-employment individuals. In addition, myelography, diskography and surgical exploration have proved degeneration or herniation in essentially all cases where these procedures have been done.

The criteria for defining the width of the lumbosacral intervertebral space we find are much more elusive. There seems to be no certain method of evaluation, unless the changes are appreciable. Measurement in millimeters is worthless, and even measurement in terms of percentage comparison to any of the spaces above has proved without merit. Our evaluation has been entirely on one's impression as to the presence of narrowing or not—which actually is an optical percentage comparison with the spaces above, modified by one's previous experience and conception of the presumed normal width. We invariably are more certain when some associated degenerative changes are present, even though they are only tiny anterior vertebral body spurs. Obviously spurs can also be present without narrowing, but the combination of the impression of narrowing plus spurs has proved reliable in those cases where proof has been obtained. The presence of the "vacuum" phenomenon (Fig. 9) or calcification is always indicative of degeneration of the disk, as is anterior planar shifting of a vertebral body demonstrable on the lateral flexion roentgenogram.

This discussion obviously cannot enter

into myelography or diskography, which are pretherapeutic diagnostic aids.

The nucleus may be extruded through the thin top or bottom plate of the contiguous vertebrae. This probably would indicate that the disk's capsule was stronger than the bony cartilaginous plates through which the herniation occurs. There is evidence that some of these intrusions may be developmental and others traumatic. An intrusion located at the level of a very limited area of backache would be suspect of being causal, if it were associated with a history of injury, especially if early and late roentgenographic examination shows the development of a dense bony jacket around the extrusion. Small Schmorl's nodules by themselves, no matter what the level, have been considered of little import. If Schmorl's nodule formation is present throughout several areas of a fairly extensive degree, the risk is presumed to be fair.

The third part of the weight-bearing mechanism is the *pedicles*, a pair of short, strong, bony shanks connecting the body of the vertebra to the fulcra or pars intermedia. They form the floor and roof of the neural foramina. It is possible that these shanks may be congenitally defective or fractured by injury. This must be rare, as it has not been observed in our entire series. Theoretically, if present it might permit the herniation anteriorly of a whole vertebral body without disturbance of the alignment of the supraincumbent spine. We have had the opportunity to see one such case elsewhere.

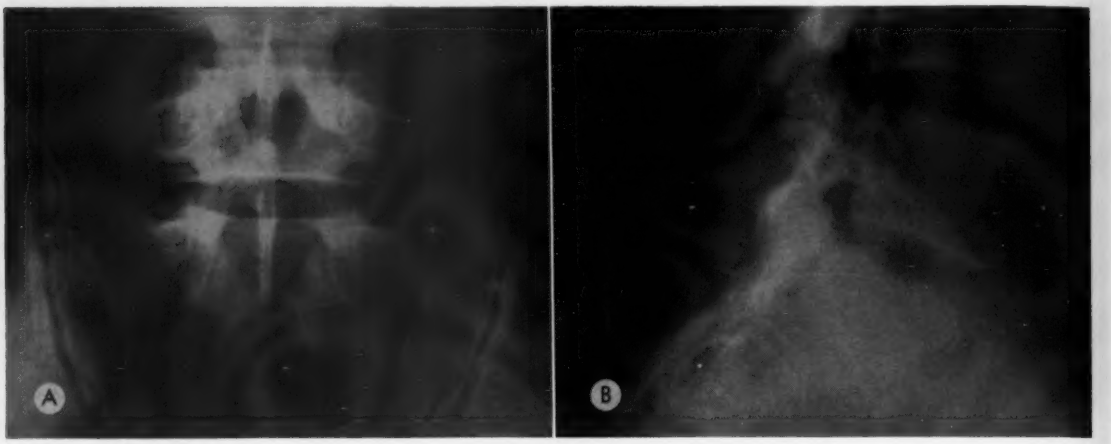
B. The force arms of the lever mechanism, as we have indicated, vary from level to level and in general consist of a posterior and paired lateral arms.

1. A spinous process is formed by the fusion of the laminae at the mid-line and their projection dorsally. Failure of fusion results in spina bifida occulta. The importance of this anomaly in all its ramifications is difficult to evaluate. Certainly the literature is of little help, as various authors express feelings at both extremes; from a normal variation at one end of the scale[12]

Fig. 9. The vacuum phenomenon is always indicative of a degenerated intervertebral disk. The demonstration of the vacuum phenomenon is more frequently seen when the lateral roentgenogram is made erect.

to an explanation for a complete paralysis on the other. Perhaps both are correct in that extensive variations of this defect occur and all with the same name. Terminology or failure to emphasize the degree of the defect as well as other associated congenital anomalies probably accounts for a good part of the divergence of opinion. In this series there is no question that spina bifida occulta (as described below) occurs much more frequently in the individuals with backache than in those without this affliction. We feel that the explanations of its effects are probably manifold. At least one may reason that the anchor for the attached ligaments and muscles is less secure and more subject to strain than if they were all attached to a solid bony strut.

Despite the lack of a complete explanation of the mechanism of action, the following criteria are applied: A small spina bifida occulta defect or failure of fusion of the lamina and the spinous processes at one level as L_5 or S_1 is presumed to be essentially a normal variation. A long defect involving the upper half of the sacrum and/or the L_4 and 5 segments or more has seemed to be significant when the "backache" and the normal series are compared. Such an anomaly places the individual in the fair risk category. Associated asymmetry of the



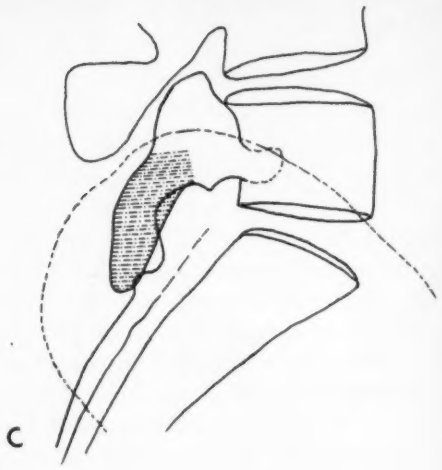


Fig. 10. "Clasp knife" deformity. (*A*) The anteroposterior roentgenogram shows the small long sacral spina bifida defect with the spinous process of L_5 dipping down and overlying this. (*B*) The lateral roentgenogram shows the spinous process to be actually folding into this area. (*C*) Diagram of the lateral roentgenogram, as it does not reproduce well. A bright light or spotlight may be necessary to see it on the usual lateral roentgenogram.

facets or mild spur formation etc. would change this to a poor risk category.

Occasionally, one sees almost the opposite condition, an anomalous overdevelopment of the spinous processes. Ferguson[4] suggests this is due to a fusion of the first sacral with the contiguous surface of the fifth lumbar spinous process to give an exaggerated hook to the caudal aspect. A normal fifth lumbar spinous process may produce the same appearance if the lumbosacral angle is exaggerated. There may be two effects: (a) In the erect position (extension) the caudal surface of this downward prolonged spinous process may be thrown into contact with the process below. This impingement is eventually manifested roentgenographically by "kissing points" of eburnation and spurs. This sclerosis is seldom seen in the young individual but, if found, it places him in the poor risk classification. (b) If there is a spina bifida occulta of the upper sacrum, the elongated or hooked spinous process of L_5 may press into the cleft, like the blade of a clasp or pocket knife folding into its handle (Fig. 10). This produces pressure on the ligamentous and cartilaginous tissues. It can occasionally compress the cauda equina, as illustrated by Gill and White.[7] We term this the "clasp knife" deformity and when present is indicative of a poor risk individual.

2. Transverse processes. The presence of one or two articulations between the transverse processes of the lowest lumbar vertebral and the sacral alae evidences a dysgenic back and creates as it were an additional strut or brace which limits and compels abnormal motion. If one or both trans-

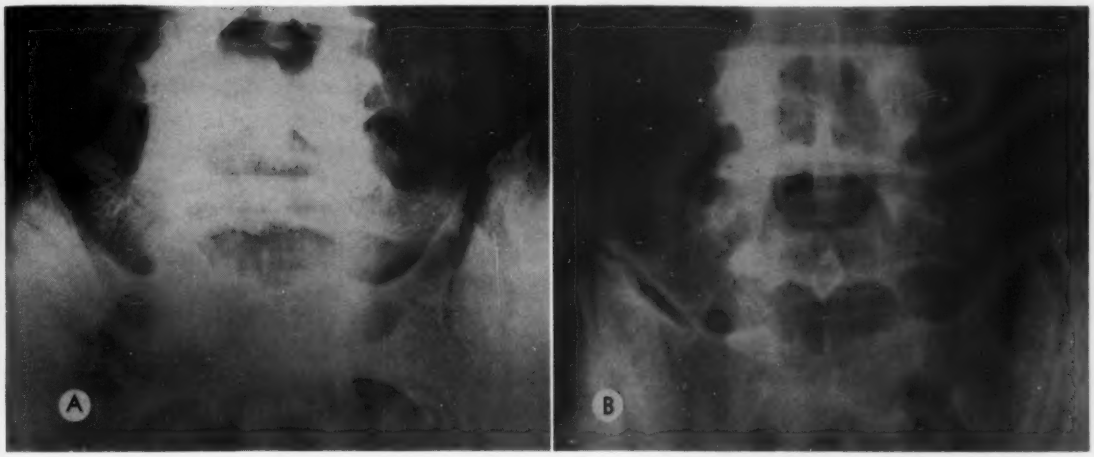


Fig. 11. Anomalous joint formation between the transverse processes of the lowest lumbar segment and the alae of the sacrum. This is frequently poorly shown on the routine anteroposterior roentgenogram (*A*) but clearly demonstrable on the anteroposterior roentgenogram with the knees up and the tube angled 20 degrees in a cephalic direction (*B*). This false joint places the applicant in the poor risk group.

verse processes are firmly fused, all motion and strain are eliminated and the level is stabilized. The associated intervertebral space is narrowed due to a lack of development and not to herniation or degeneration of the disk. Though this individual is perhaps more prone to develop disk disturbances at a higher level, the degree of risk in our experience has been too low to be determined with any certainty. Stinchfield and Sinton[15] find the incidence of this disk disturbance to be high, though they did not seem to separate the solid or fused transitional cases from those with a transverse process arthrosis. Perhaps this could account for their differing opinion. In our series those with an anomalous arthrosis have been highly significant. To identify these articulations, a knees-up cephalic angulation roentgenogram is required. The ordinary anteroposterior roentgenogram may fail completely to demonstrate this abnormal joint (Fig. 11 and 12). Eburnation of the articulation and spurs are commonly seen, which by itself tends to point out their importance. Flexion lateral roentgenograms may show motion, which also confirms the presence of a movable joint. The latter is usually not too helpful, as even when obvious joints are present, limitation of motion is so marked that the change is hard to detect.

The finding of unilateral or bilateral articulations between the transverse processes of the lowest lumbar segment and the alae of the sacrum is cause for classification as a poor risk. If one or both sides are solidly fused, the anomaly is considered to be without significance.

Fractures of the transverse processes, if ununited and showing marginal sclerosis, may betoken a hazard. Multiple old fractures would probably indicate hemorrhage in muscles and possibly restricted motion, requiring further physical examination.

Fig. 12. Multiple anomalous joints involving the transverse processes in relationship to each other as well as the ala of the sacrum. A poor risk classification.

IV. GENERALIZED CONDITIONS

Scoliosis has not been a particular problem. In a few cases with lumbar components there has been rotation, wedging or spurring of the vertebral bodies and they have been rated as a poor risk. Minimal or mild scoliosis without wear and tear phenomena would call for a fair risk classification.

Spinal surgery, evidence of previous myelography as manifested by the presence of opaque oil, generalized demineralization, evidence of previous inflammatory disease as tuberculosis or osteomyelitis, Marie-Strümpell spondylitis, and similar phenomena carry a poor risk classification.

In this series sclerosing iliitis (osteitis condensans ilii) has not been present in the young pre-employment male. As elsewhere in the literature, we have seen it frequently both in the young female patient complaining of backache as well as an incidental finding in persons without symptoms. We would feel obligated to presume a poor risk classification if it appeared in the pre-employment individual.

Occasional other abnormalities are uncovered, such as renal calculi, aortic arteriosclerosis, aneurysms, etc. These are weighed and related to the over-all physical examination.

DEGENERATIVE CHANGES

It is our feeling that degenerative changes as manifested by spurs about the vertebral bodies, in the zygapophysial facet areas, about the crests of the ilia, and sacroiliac synchondroses are representative of wear and tear. Though the prognostic significance is undoubtedly difficult to evaluate in a forty-five or fifty year old individual, the finding of such changes in the young adult has an entirely different connotation and is indicative of a predisposition to degenerative phenomena. Indeed, it may be the only evidence of an unstable or poor risk back. A few small anterior vertebral body spurs in the upper lumbar spine are of elusive importance, and are ignored when seen as isolated phenomena. Such findings, however, in the lumbosacral region would place the individual in the fair risk category; and if they are moderate or extensive, in the poor risk group. The demonstration of roughening of the crests of the ilia or spurs at the inferior sacroiliac synchondrosis are considered in the same light. A point of caution is to be made about identifying spurs at the inferior portion of the sacroiliac synchondrosis, as the normal appearance will occasionally mimic this picture.

COMMENT

It is to be reiterated that the critical area of concern lies at the L_{4-5} and lumbosacral level. Minor changes here carry an important connotation that would be ignored elsewhere. It is also to be borne in mind that there are various degrees and combinations of the abnormalities and particularly their differing significance in the various age groups. Intelligent thought and keen medical judgment must temper the use of these criteria. As here applied, the risk classifications refer to the prognosis in the future five, ten or even twenty years. The absence of current or past backache symptoms does not modify one's judgment. The strong supple muscles and ligaments of youth give a protection that will not be present in later years, when there is loss of elasticity and turgor of the connective tissues plus the accumulation of the inevitable multiple insults of life and labor.

The great majority of our reasons and criteria for classifying individuals into the three risk categories are rather certain and self-evident. It is also obvious that no general agreement of all the criteria could be had nor even anticipated. On the contrary, a good deal of divergent opinion and comment is expected. After long use and many years of association, the three authors will differ on some cases, though such differences are becoming less and less frequent. Needless to say, over the years of evolution of this project the criteria have been continuously modified, and the future shall

undoubtedly bring further changes. Our detailed methods have been given in the hope that others will not only find them useful, but also will be better able to present additional evidence and modifications.

SUMMARY

On the basis of roentgenographic studies of the lumbosacral spine, pre-employment applicants are graded as good, fair and poor risks. The fourth and fifth lumbar, and first sacral segments are of critical interest. Irregularities or abnormalities here are considered of high importance. This is particularly so in the younger applicants under thirty years of age where every sign of stress, wear and tear is considered significant. The following general criteria are applied, though sensible medical judgment modifies the decision in each.

A. *Good risk* (employable for any job requiring physical strain or effort).
   1. Normal findings.
   2. Atavistic changes.
   3. Limbus body.
   4. Four or six lumbar vertebrae.
   5. Ununited epiphysis of the pars articularis.
   6. Mild asymmetry of the zygapophysial facets.
   7. Small spina bifida occulta.
   8. Schmorl's nodules in upper lumbar spine.
   9. Transitional lumbosacral vertebrae with solid fusion of one or both transverse processes.
   10. "Increased" lumbar lordosis.

B. *Fair risk* (defects present of such a nature as to permit a person's employment for labor, but not for repetitious or continued heavy low back work).
   1. Appreciable asymmetry of the zygapophysial facets.
   2. Moderate spina bifida occulta.
   3. Schmorl's nodules at the L4 or L5 level.
   4. Moderate degenerative changes in the upper lumbar spine, or small vertebral body spurs in lower areas.

C. *Poor risk* (defects of such a magnitude as to render heavy lifting or labor inadvisable).
   1. Spondylodysgenesis (spondylolysis) or spondylolisthesis.
   2. Transitional lumbosacral vertebrae having transverse process articulations with the ala of the sacrum.
   3. Thin intervertebral disk space at the L4–5 or lumbosacral level.
   4. Marked general hypertrophic degenerative changes in the upper spine, or moderate if at the L4, L5, or S1 level, and mild if at this lower level and in a young applicant.
   5. Wedged vertebra.
   6. Hemangioma or neoplasm of any form.
   7. Extensive spina bifida occulta.
   8. "Clasp knife" deformity.
   9. Combinations of other changes, as asymmetry of zygapophysial facets plus degenerative changes.
   10. Evidence of previous surgery, myelography, previous infection, Marie-Strümpell spondylitis, systemic disease as osteoporosis, other gross congenital anomalies as hemivertebra, etc.

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ROENTGENOGRAPHIC ABNORMALITIES IN SOLDIERS WITH LOW BACK PAIN: A COMPARATIVE STUDY*

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LOW back pain has been, at one time or another, attributed to almost every bony abnormality which can be visualized roentgenographically in the low back. The significance of these various abnormalities is still not settled. Their presence has sometimes been considered to be an indication for surgery, particularly spinal fusion. Since World War II emphasis has been placed on the bony structure of the low back in pre-employment examinations for laborers; routine low back roentgenograms are taken by some firms prior to employment,[9] and employment refused on the basis of the abnormalities found on roentgenographic examination.

What are the roentgenographic findings in a normal low back? To answer this question a detailed roentgenographic study[1] was made of 100 soldiers who had never suffered an injury to the back and who had never had backache. With few exceptions these men had been subjected to strenuous military training and some had been in combat. The roentgenograms which were obtained in each of the subjects consisted of: (1) routine views of the lower lumbar spine and sacrum in anteroposterior and lateral projections; (2) a roentgenogram made with the roentgen tube tilted cephalad at a 30 degree angle to show the sacrum, sacroiliac joints and the relationship of the transverse processes of the last lumbar segment to the lateral masses of the sacrum; (3) roentgenograms made in right and left semilateral projections with the subject rotated 30 to 45 degrees, for visualization of the laminae of the vertebral arches and the articular processes with their facets. All of the roentgenograms were studied by each of us. In cases where there was any doubt as to the nature of an abnormality, stereoscopic roentgenograms and roentgenograms made in other projections were obtained for further study. The findings of these normal backs are summarized in Column 1 of Table 1. Only 20 spines showed no bony abnormalities.

The present study is a companion study concerned with the roentgenographic findings of a parallel group of soldiers who presented themselves consecutively for treatment because of back pain at the lumbar and sacroiliac levels.

MATERIAL

Two hundred male soldiers between the ages of nineteen and fifty-one with an average age of 30.4 years were included in this group. The average age of the men in the control group was 26.6 years. Practically all of these men had been subjected to strenuous military training and many had been in combat.

For the purposes of this study, no attempt was made to correlate the symptoms with the roentgen findings. The cases were examined consecutively and the variations in the roentgenograms of each soldier were studied and tabulated in conference. A summary of the principal variations found in this series is shown in Table 1, Column 2. For comparison, the roentgenographic findings in the control group are shown in the same table.

TECHNIQUE OF EXAMINATION

The technique of examination was identical to that used in the control series as outlined in the second paragraph of this paper. Where necessary, stereoscopic roentgenograms in oblique projections and special projections were obtained for more detailed study.

* Study was made during a period of military service at Bruns General Hospital, Santa Fe, New Mexico.

TABLE I

SUMMARY OF VARIATIONS

Variations	Control Group (100 spines) Per cent	Symptomatic Group (200 spines) Per cent
No Roentgen Variations	20	20
Spina Bifida Occulta	36	39
Transitional lumbosacral Vertebrae	11	10.5
Asymmetric Facets	39	25.5
Narrowing of Lumbosacral Joint	11	17
Degenerative Arthritis of Lumbosacral Joint	4	7
Sacroiliac Arthritis (degenerative sclerotic)	1	2
Sacroiliac Arthritis (rheumatoid ankylosing)	0	1.5
Increased Acuity of Lumbosacral Angle	2	5.5
Spondylolysis	1	5.5
Spondylolisthesis	5	9
Ununited Epiphyseal Centers	2	2.5
Generalized Osteoporosis	1	0

It should be commented that the oblique (semilateral) projection was found to be essential for the diagnosis of neural arch defects in a number of instances. Neural arch defects could be seen in the lateral views of the lumbosacral region in less than one-third of the cases, where they could be shown in the oblique projections. In none of the cases could the diagnosis of spondylolysis be made with certainty by inspection of the anteroposterior roentgenogram alone.

SPONDYLOLYSIS AND SPONDYLOLISTHESIS

Forward displacement of a vertebra (spondylolisthesis) occurs most frequently at the fifth lumbar vertebra upon the sacrum. Occasionally, the fourth lumbar segment and still less frequently other vertebrae may show this displacement. The most common underlying predisposing factor preceding spondylolisthesis is a bilateral defect in the neural arch in the region of the pars interarticularis (spondylolysis). Another cause for forward displacement of the vertebra has been described by Goldthwait[8] and Müller,[7] namely, faulty development of the inferior articular process. In one case of spondylolisthesis in this series (Fig. 1), the underlying cause of the slipping was a destruction of the inferior apophyseal joint space which permitted the body of the fifth lumbar vertebra to glide forward on the sacrum and to rotate slightly. The routine roentgenograms showed that the left lumbosacral apophyseal joint was obliterated and that marked sclerosis of the joint margins had taken place (Fig. 2). Junghanns[4] has described a similar condition with a reverse slipping as the result of chronic inflammatory changes in the joints.

The incidence of spondylolysis and spondylolisthesis in soldiers has been reported by other investigators. Galluccio[2] found this abnormality in 10 per cent of 142 routine lower spine examinations. Meschan[5] examined 1,131 lumbosacral spines of soldiers in an overseas hospital and found the incidence of spondylolysis 5.1 per cent. Of these 72 per cent showed evidence of spon-

FIG. 1. An unusual type of spondylolisthesis encountered in this series. Lateral roentgenogram shows a Grade 1 spondylolisthesis.

dylolisthesis. The author does not state how many of these men complained of backache. The incidence of spina bifida occulta in this group was 25.9 per cent and of transitional vertebrae 10.4 per cent. Raney[8] who examined 300 patients because of low back pain by means of routine and oblique roentgenograms reported an incidence of isthmus defects of 31 per cent.

There were 29 occurrences of spondylolisthesis in this study as compared with 6 in the control group. Of these there were 28 cases of true spondylolysis. Twenty-four showed bilateral defects in the region of the pars interarticularis of the fifth lumbar vertebra and four showed unilateral defects. Of the 28 with spondylolysis, 17 had an associated spondylolisthesis. Sixteen were of Grade I, and one was of Grade II severity, according to the Meyerding[6] classification.

Of the 28 cases showing a spondylolysis, 14 had an associated spina bifida occulta of the first sacral segment; an incidence of 50 per cent as compared with 39 per cent for the entire group. A similar incidence was encountered in the control group. Meyerding[6] has reported an incidence of 35 per cent of spina bifida occulta in association with spondylolisthesis.

Fig. 2. Oblique roentgenogram shows obliteration of the lumbosacral apophyseal joint and marked sclerosis of adjacent bone. With destruction of the joint, the fifth lumbar vertebra was displaced forward on the sacrum.

In the 29 cases of spondylolisthesis found in this group, degenerative arthritis occurred in 6.9 per cent; narrowing of the lumbosacral intervertebral joint space in 13.7 per cent; increased acuity of the lumbosacral angle in 6.9 per cent and rounding of the anterior margin of the sacrum in 20 per cent.

LUMBOSACRAL AND SACROILIAC JOINTS

Degenerative arthritic changes in the lumbosacral and sacroiliac joints were found in 9 per cent of the cases as compared with 5 per cent in the control group. The average age in this group was forty-one years. This finding is consistent with the fact that beginning at about the age of thirty-five, osteoarthritis is seen with increased frequency with advancing years. Three of the symptomatic group presented moderate to advanced changes of rheumatoid arthritis of the sacroiliac joints. The average age was twenty-two years.

Narrowing of the lumbosacral intervertebral joint space was found in 17 per cent of the cases as compared with 11 per cent in the control group. An increase in the acuity of the lumbosacral angle was encountered in 5.5 per cent of the patients as compared with 2 per cent in the control study.

SPINA BIFIDA OCCULTA AND TRANSITIONAL VERTEBRAE

The incidence of congenital variations, such as spina bifida occulta and transitional lumbosacral vertebrae was found to practically be the same in both the control series and the symptomatic group. Spina bifida of the first sacral segment occurred more frequently when there was an associated spondylolysis (50 per cent).

COMMENT

The number of neural arch defects (spondylolysis) in the spines of soldiers complaining of backache was found to be two and one-half times as high as in the control group. The differences in other variations, such as degenerative arthritis of the lumbosacral joint and sacroiliac joints, narrowing of the lumbosacral intervertebral joint space and increased acuity of the lumbosacral angle were less striking. The percentage of spines showing no variation whatsoever was the same in both groups. Likewise there was little difference in the incidence of such congenital anomalies as spina bifida occulta and transitional vertebrae in the control and symptomatic group.

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THE "SWAYBACK" SYNDROME*

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THE purpose of this paper is to describe the diagnostic features of a little discussed roentgenologic entity which properly may be called the "swayback" syndrome.

HISTORIC REVIEW

A review of the literature indicates that the term "swayback" has not been applied previously to the set of roentgenographic findings comprising this entity. Baastrup[1] delineated findings in the lumbar spine which have since been referred to as "Baastrup's disease" (1936). His description contains some of the features encountered in the "swayback" syndrome. Baastrup emphasized the role of lumbar spinous processes which become contiguous because of an increase in the lumbar lordosis and form interspinous arthritic changes. He felt that this entity was encountered more frequently in males engaged in heavy labor. In such individuals, Baastrup postulated a sudden increased lordosis resulting in contiguous spinous processes with injury to the interspinous soft tissues, including the ligaments and musculature. He described the resultant development of osteoarthritis between the spinous processes with cartilage destruction and impingement of adjacent bone margins followed by reactive osteosis, subchondral cysts, osteophytes, joint mice and ultimately, ankylosis. In some instances, Baastrup felt that articular facets (pseudoarthrosis) may form between the contiguous spinous processes.

In 1949, Faulong, Leger and Akhras[2] referred to Baastrup's description and indicated that "lumbalgia" could be due to Baastrup's disease.

In 1954, Salvini[3] emphasized that "Baastrup's disease may occur in asymptomatic individuals without any occupational background or heavy labor." He also indicated that this entity may occur in both males and females. As far as we can tell, these three references constitute the only literature which deals even superficially with some of the aspects of the "swayback" syndrome.

CLINICAL MATERIAL

The material collected was obtained during the course of the routine interpretation of roentgenograms during the past year in a busy Department of Radiology in a general hospital. During this period, a total of 56 cases was collected with the classic and characteristic features of this syndrome. These cases were unselected and consecutive and included patients sent to us for skeletal surveys with no back pain. Of the 56, adequate clinical records on 40 were available.

There were 34 females and 6 males. The ages ranged from forty-two to eighty-seven years with an average age in the range of sixty-four years (Table 1).

ROENTGEN CRITERIA

The diagnosis of the "swayback" syndrome is suggested clinically to the orthopedic surgeon but is best made with certainty by the radiologist. The roentgen criteria consist of the following:

1. Increase in lumbar lordosis with an ac-

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Table I

No. of Cases	Sex	Age Range	Average Age
		(yr.)	(yr.)
34	F	42–87	63.3
6	M	44–75	65.7

companying roundback deformity in the dorsal spine.

2. Closely situated or contiguous spinous processes in the lumbar area.

3. Eburnation about the spinous processes, particularly in the later stages with secondary bursal involvement.

4. Impingement of the inferior articular facets on the laminae below, with eburnation and marginal sclerosis about these sites.

5. Small joint arthritis manifested by destruction of the cartilage and marginal eburnation, as well as exostosis formation.

6. Disk cartilage thinning.

7. Misalignment of the lumbar centra, particularly in the advanced stages.

8. Paucity of anteriorly situated exostoses involving the lumbar centra.

We have chosen to classify the roentgen findings into three major groups: mild, moderate and severe. In our series, there were 12 mild, 11 moderate and 17 severe cases. In the mild form, we included those cases which showed the characteristic postural changes of an accentuated lumbar lordosis with the spinous processes in the lumbar spine close but not necessarily contiguous. In the moderate group, there were increase in the lumbar lordosis with the spinous processes contiguous with or without eburnation, beginning impingement of the inferior articular facets on the laminae below and early small joint arthritis. In the severe form, we found considerable increase in the lumbar lordosis with contiguous spinous processes showing peripheral eburnation, varying degrees of impingement of the articular facets upon the laminae below with surrounding eburnation, definite small joint arthritis, degenerative disk disease and varying degrees of misalignment (see Table 1). An almost uniform finding was a remarkable paucity of exostoses involving the anterior aspects of the lumbar centra. Table II represents a summation of the roentgen findings in the three degrees of "swayback."

Table III

BACK PAIN AND DEGREE OF "SWAYBACK"

Degree of "Swayback"	Asymptomatic	Backpain
Mild	12	5	7
Moderate	11	7	4
Severe	17	8	9
Total	40	20	20

The diagnostic features are quite apparent in routine frontal and lateral roentgenograms of the lumbar spine. Oblique roent-

Table II

ROENTGEN FINDINGS IN "SWAYBACK" SYNDROME

Degree of Swayback	Increased Lumbar Lordosis	Contiguous Spinous Processes	Impingement of Inferior Articular Facets on Laminae Below	Small Joint Arthritis	Disk Degeneration	Misalignment
Mild	X	X	O	O	O	O
Moderate	XX	XX	X	X	X	O
Severe	XXX	XXX	XX to XXX	XX to XXX	XX	XX to XXX

Symbols:

O—generally absent.
X—mild.
XX—moderate.
XXX—severe.

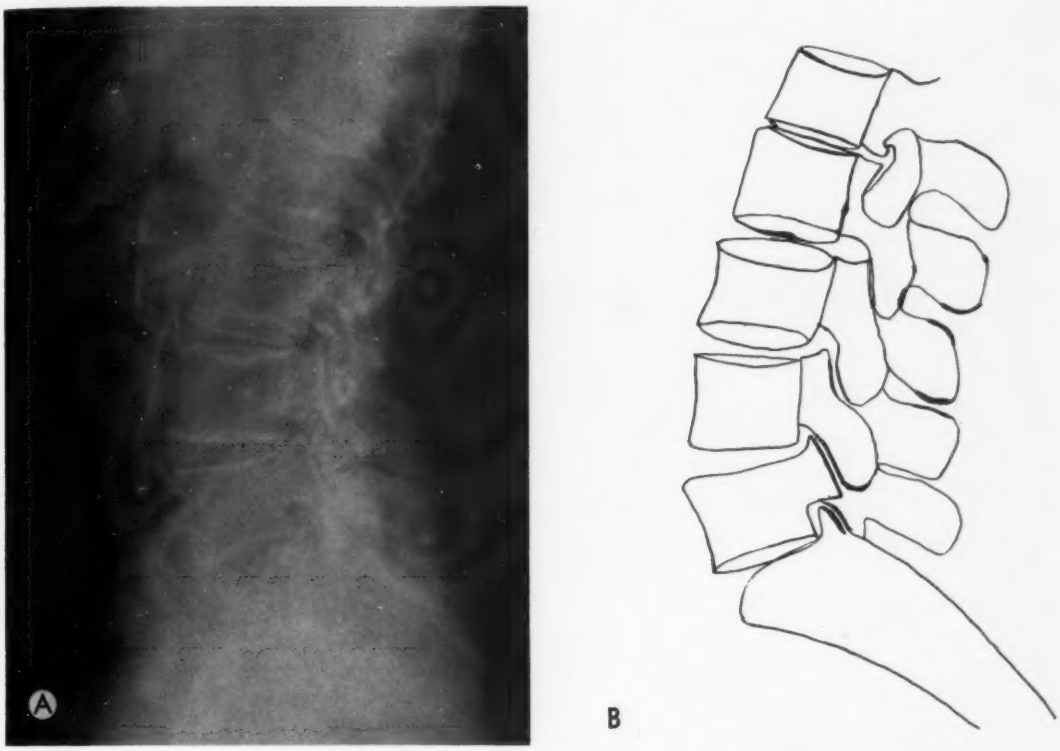

Fig. 1. (*A* and *B*) Lateral roentgenogram of the lumbar spine with line drawing in "mild" case. There is a greater than average lumbar lordosis with the lower lumbar spinous processes quite close and almost contiguous.

genograms may be added to further delineate the changes in the posterior small joints and the relationship between the inferior articulating facets and the laminae. In our experience, these oblique roentgenograms usually are not necessary.

Figures 1, *A* and *B;* 2, *A* and *B;* and 3, *A* and *B* are representative roentgenograms with related line drawings for the mild, moderate and severe groups. Figure 4, *A* and *B* demonstrates the findings in the oblique views (same case as shown in Figure 3, *A* and *B*).

SYMPTOMATOLOGY

It has been our impression that the "swayback" syndrome is generally responsible for symptoms of varying degrees of back pain, oftentimes quite disabling. An attempt was made in this study to determine the frequency of significant back pain in patients with "swayback" findings and to relate the various grades of "swayback" roentgenologically to the severity of symptoms. Table III shows that there is no accurate correlation between severity of the roentgen findings and occurrence and severity of back pain. Half of the patients examined complained of back pain in varying degrees.

An attempt to relate the physical findings to the roentgenologic features was not contributory.

DISCUSSION

Although this report is of a preliminary nature, the "swayback" syndrome is a real roentgenologic entity. Individuals showing the characteristic roentgen features of this syndrome may suffer from varying degrees of back pain as a result of the changes described, while others may be asymptomatic. However, in spite of this lack of positive correlation, the importance of this entity should not be discounted. The roentgen findings of an increase in lumbar lordosis,

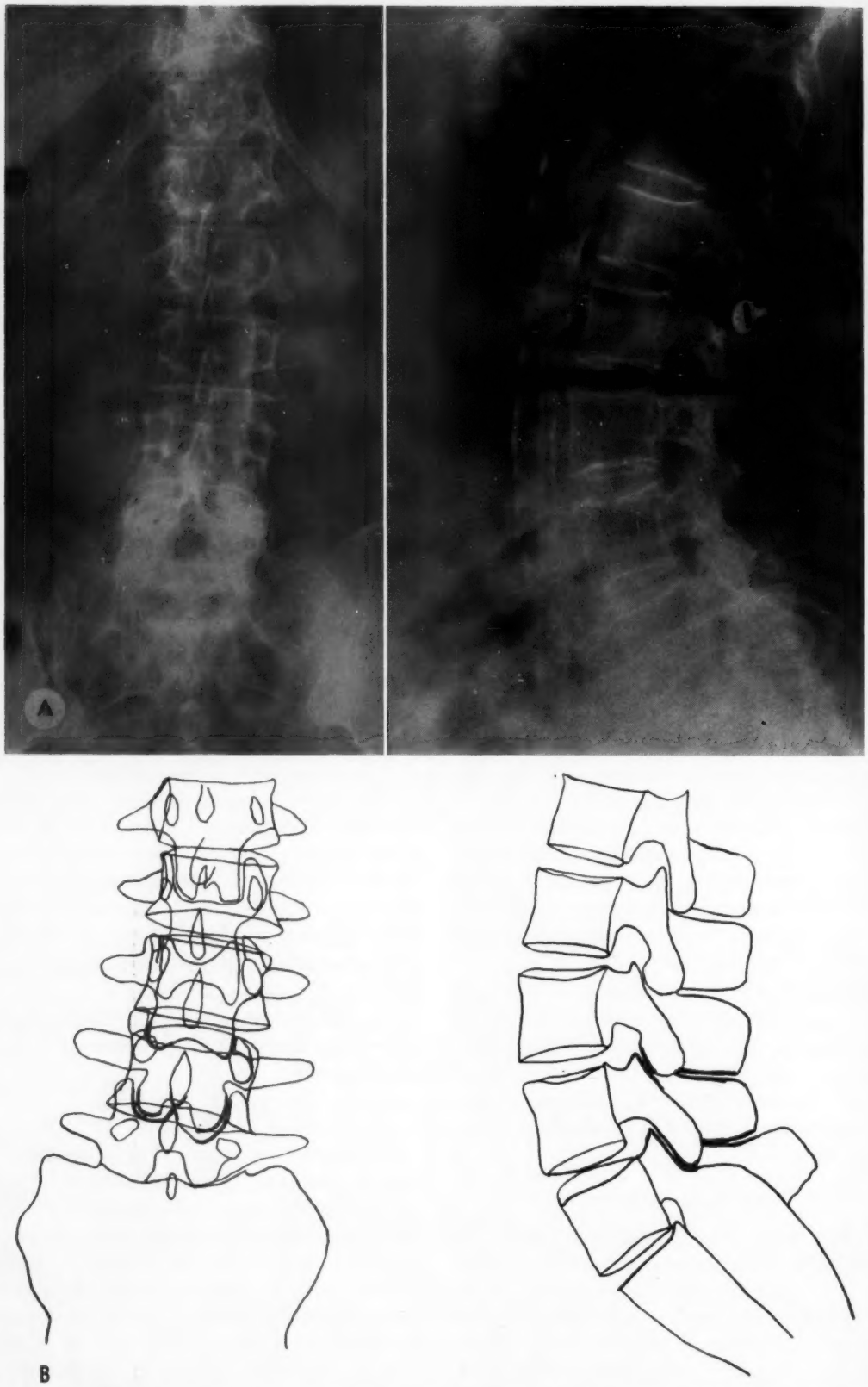

FIG. 2. (*A* and *B*) Anteroposterior and lateral roentgenograms of the lumbar spine with line drawings in "moderate" group. The lumbar lordosis is further accentuated, the spinous processes in the lower lumbar area are not contiguous with early surrounding eburnation, the inferior articulating facets have begun to impinge on the laminae below with adjacent sclerosis and early small joint arthritis is present.

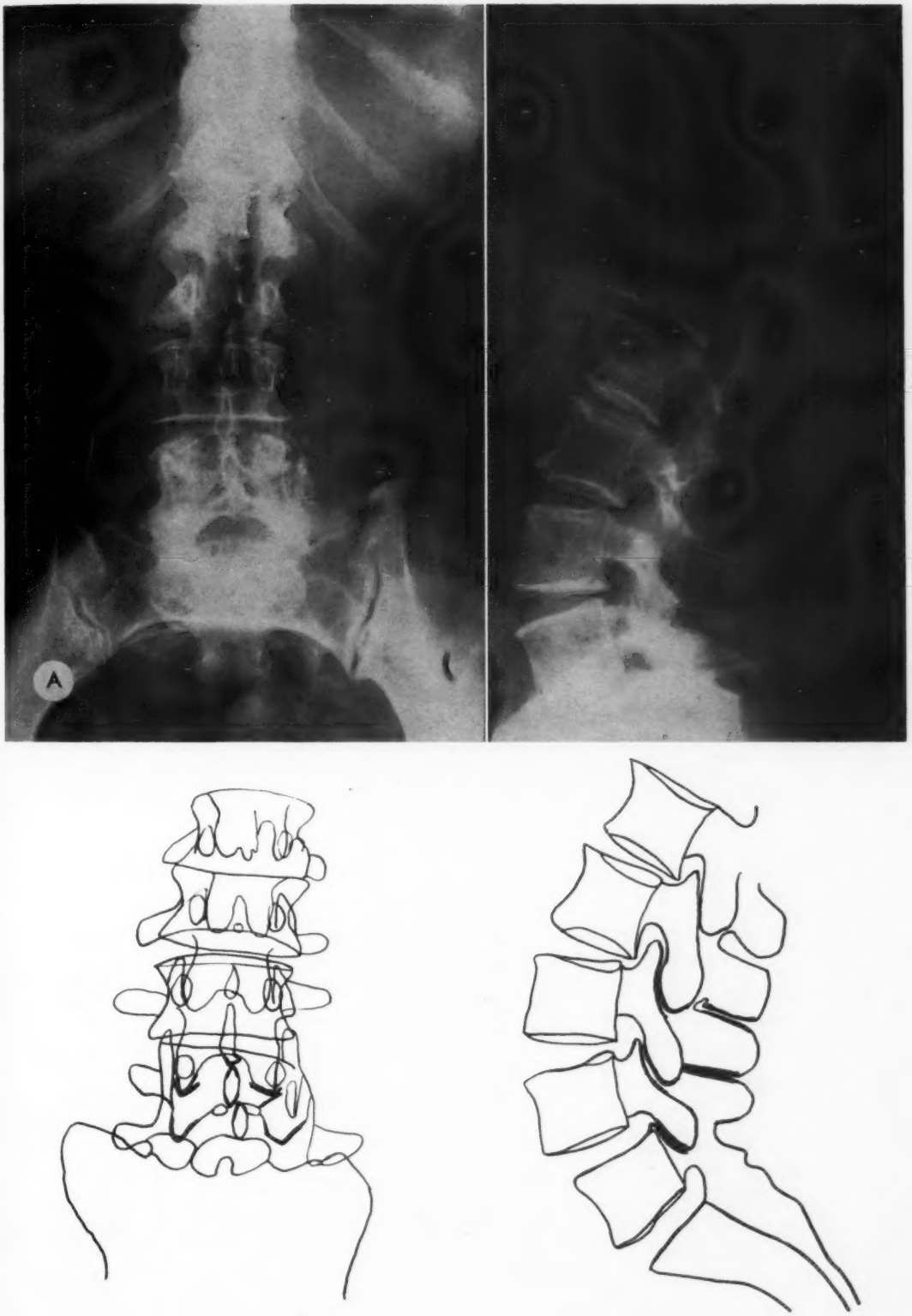

FIG. 3. (*A* and *B*) Anteroposterior and lateral roentgenograms of the lumbar spine with line drawings in "severe swayback." The lumbar lordosis is considerable, the spinous processes are contiguous with marginal changes, the inferior articulating facets impinge on the laminae below with peripheral eburnation, there is definite small joint arthritis and there is disk cartilage degeneration at L4–L5 with misalignment at this site. Note the relative lack of exostoses of the anterior margin of the lumbar centra.

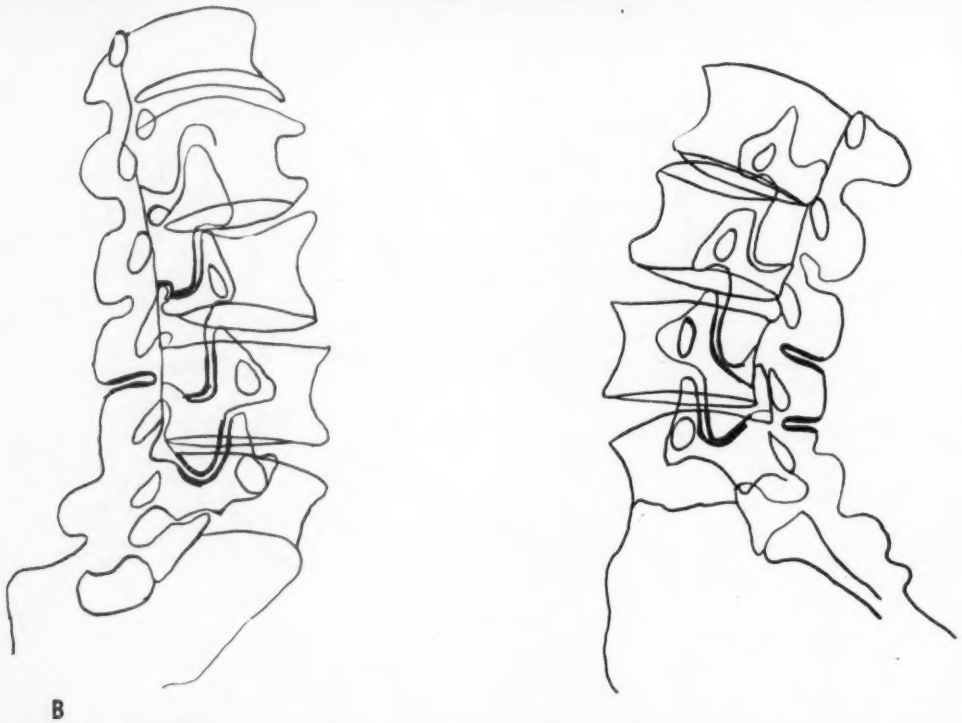

FIG. 4. (*A* and *B*) Oblique roentgenograms with line drawings of same patient described in Figure 3. The oblique views may be helpful but are not usually necessary.

contiguous spinous processes, inferior articular facets impinging on the laminae below, with small joint arthritis and resultant degenerative disk disease and misalignment, comprise a definite entity. The accurate description and evaluation of these findings by the radiologist will prove of service to the clinician in a number of instances where the cause of back pain might otherwise go unexplained. The radiologist's role in detecting this entity is manifestly important.

SUMMARY AND CONCLUSIONS

An entity which we have called the "swayback" syndrome is presented. The roentgen features of this syndrome are described and illustrated and a simple classification is given.

An attempt has been made to correlate the incidence and severity of low-back pain to this specific set of roentgen findings; no accurate correlation is noted.

The role of the radiologist in diagnosing this entity is emphasized.

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TENNIS ELBOW: A RE-EVALUATION*

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THE first description of the so-called tennis elbow syndrome is attributed to Bernhardt[1] (1896), who considered it an occupational neuralgia. This concept, however, is not correct; nor is the name tennis elbow a proper term, because among those who suffer from this disease only a few are tennis players. Nevertheless, this is probably the most commonly used name, though in the literature the synonyms epicondylitis, radiohumeral bursitis, epicondylalgia and golf elbow may also be found. The respective literature, having been reviewed by Cryiax[7] and more recently by Christopher,[3] will not be discussed here.

The name tennis elbow and its synonyms designate a clinical syndrome of the elbow, characterized by pain and local tenderness of variable degree and sometimes swelling and redness—in the region of the lateral epicondyle of the humerus. Limitation of motion is commonly present. The pain is generally precipitated by active contraction of the extensor muscles of the forearm or by passive stretching of their common tendon which is inserted at the lateral epicondyle of the humerus. In active contraction of the muscle, the pain is more marked if effort is made against resistance. Such resistance may be the action of gravity, *e.g.*, an attempt to lift a heavy object while the elbow is extended and the forearm is in pronation. Passive stretching of the tendon is an important diagnostic method in the hands of the examiner. It can be brought about by forced pronation of the forearm and flexion of the hand and wrist, while the elbow is being held in extension. Sometimes there is a distinct feeling of resistance during such procedure.[17] If the patient indicates pain in the region of the lateral epicondyle during this maneuver, the diagnosis is established.

The etiology and pathogenesis of this condition is obscure and so far no agreement has been reached as to the nature of the underlying pathologic changes. The following is a list—probably incomplete—of the pathologic changes suggested: bursitis, adventitious bursitis, tear of the muscular attachment of the extensor carpi radialis brevis muscle with subsequent periostitis, fascial tear, myofascitis, periostitis traumatica, arthritis of the radiohumeral joint, synovitis traumatica, and deranged fringes of the synovia. Osgood's[18] concept that a bursa (radiohumeral or Osgood's bursa) is the primary location of the disease has been favorably received by many. The suggested therapy of this disease is generally approached through the concept regarding the underlying pathology. Consequently, the methods of therapy described in the literature are as varied as the concepts of the disease itself. Good results have been reported by different physiotherapeutic methods and conservative orthopedic manipulations, as well as by surgery, both with and without opening of the joint capsule.

Since Schmitt's[21] report in 1921, it has become increasingly evident that calcium salt deposition at the lateral epicondyle of the humerus may be associated with the syndrome under discussion. Similar observations have been published by Carp,[2] Hamilton,[11] Crawford,[6] Hughes,[13] and Cowan and Stone.[5] Because of the similarity of the clinical symptoms and the roentgenographic findings of calcification, the possibility of a relationship between tennis elbow and the entity known as bursitis, tendinitis, peritendinitis, or periarthritis calcarea* was suggested. Periarthritis cal-

* The name periarthritis calcarea has been suggested by the author[9,10] and will be used in this paper.

* From the Dispensary of the United States Army, the Pentagon. This is a part of the paper "Observations on Periarthritis Calcarea and a Re-evaluation of the Tennis Elbow Syndrome" presented at the Eastern Conference of Radiologists in Washington, D. C., March 12, 1954.

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carea occurs most commonly in the shoulder region, but has also been reported in almost all of the joints of the extremities (Sandström,[20] Hitchcock,[12] Gondos,[9,10] and others). The disease is a self-limited one and the calcification diminishes in size or disappears within a few weeks in a majority of cases.

The purpose of this paper is to show that tennis elbow does not represent a separate entity but that it is identical with periarthritis calcarea and should be grouped under this heading. This concept has been developed on the basis of clinical and roentgenographic observation of 20 cases of tennis elbow in the Dispensary of the United States Army, The Pentagon, 7 of these cases displaying roentgenographic evidence of calcium salt deposition at the lateral epicondyle of the humerus. We were impressed by the similarity of both diseases with regard to the clinical signs and symptoms as well as the roentgenographic manifestations (Fig. 1 and 2). In a systematic review of our cases, every attempt to differentiate the two conditions, either clinically or roentgenographically, was unsuccessful. In retrospect, it became evident that the two terms had been used interchangeably, depending mainly upon the method by which the diagnosis was approached. When more emphasis was placed on the roentgenographic examination, the disease was called periarthritis calcarea. In instances when only clinical examination was performed or when the roentgenogram did not reveal evidence of calcium salt deposition, a diagnosis of tennis elbow was made. Other authors reporting on this subject apparently handled the question of terminology with a similar unconscious mental attitude.

In order to further evaluate this concept, we reviewed the respective literature on pathology to which partial reference has already been made. We also present two statistical studies of our clinical material and a roentgen-anatomic study of the elbow.

Recently, Spencer and Herndon[22]—on reporting 49 operated cases of epicondylitis—found that "the deep fascia (just distal to the epicondyle) and tendinous origin of the muscles from the epicondyle frequently appeared somewhat thicker and denser than usual." In 2 cases, small calcium salt deposition in the tendinous attachment was found. Though these authors believe that the underlying pathology is traumatic periostitis or myofascitis, with which we cannot agree, this report is interesting because it localizes the disease in the tendinous attachment of the common extensor muscles. The pathologic substrate of the tennis elbow was investigated histologically by Meherin and Cooper.[16] They operated on 9 such cases and found gross calcification in 2. Removed parts of the extensor tendon, including periosteum scraped off from the lateral epicondyle, were examined histologically. Some of the sections showed degeneration and "many of the sections showed depositions of small amounts of calcium.

Fig. 1. Calcium salt deposition adjacent to the lateral epicondyle. The patient displayed symptoms and signs of tennis elbow.

Fig. 2. (*A*) Faint calcific density adjacent to the lateral epicondyle. The patient had acute pain and tenderness in the area of the lateral epicondyle. (*B*) Three weeks later most of the calcification has disappeared; the patient was free from symptoms at this time.

. . . These were too small, too few and too scattered to have been detected by x-ray examination." They found no evidence of inflammatory process in either the tendon, periosteum or muscle, and there was no evidence of partial tear of either tendon or muscle. The pathologic observations referred to are essentially the same as found in periarthritis calcarea in the shoulder region and elsewhere. Since Codman's[4] intensive study of the shoulder, we know that the bursa, if involved, is almost always secondary to an involvement of the tendon, though other periarticular structures also may be primarily involved. Too, it has become known that the pathologic process is primarily a degenerative one (Codman,[4] Pedersen and Key,[19] etc.). Calcification demonstrated roentgenographically is a characteristic manifestation of this entity. The calcium salt deposition in anatomic or surgical observations is commonly found to be small or microscopic, in an otherwise normal appearing tendon. In DePalma's[8] study of 100 shoulders in autopsy cases "microscopic deposits of calcium were encountered more frequently than macroscopic deposits."

Though the question still remains controversial in the literature, these pathologic studies suggest that in tennis elbow the localization of the disease is mostly in the tendon and the nature of the process is a degenerative one associated with calcium salt deposition. The similarity to the characteristics of periarthritis calcarea is noticeable. It is also obvious that in a number of cases of painful elbow diagnosed as tennis elbow, and in a number of painful shoulders clinically diagnosed as periarthritis calcarea, the roentgenograms may not reveal calcification because of the minute deposits of calcium salts. This is the reason why a diagnosis of periarthritis calcarea may be made without roentgenographic evidence of calcium salt deposition.

The relationship of calcium salt deposition to the clinical symptoms and signs is

obscure. These are manifestations of an associated inflammation which may in acute cases show all of the requirements of Celsus and Galen: calor (heat), rubor (redness), dolor (pain), tumor (swelling), and functio laesa (impairment of function). Calcification is, however, relatively commonly found without any evidence of inflammation, indicating that the calcium salt deposition cannot be responsible, or at least not alone, for the clinical symptoms and signs. We have followed a patient suffering from symptoms of tennis elbow and showing calcification at the lateral epicondyle which has remained entirely unchanged for four and one-half months. The pain and other symptoms, however, were absent after ten days. In another case the patient displayed a hazy calcification with a dense calcific center adjacent to the lateral epicondyle. The patient had acute pain, swelling, local tenderness and impairment of function, all of which subsided after seven days. Roentgenographic examination half a year later revealed disappearance of the hazy calcification but the dense calcification previously seen in the center remained unchanged. The patient was asymptomatic at this time.

It seemed to be of interest to correlate the incidence of visible calcification in the tennis elbow syndrome and periarthritis calcarea. If they are identical diseases as postulated, a similar percentage involvement may be expected.

The respective statistical study includes a two-year period (May 1, 1951–April 30, 1953) in the Dispensary of the United States Army, The Pentagon. During this interval, 20 such cases of painful elbow were seen, in which a clinical diagnosis of tennis elbow or of its equivalent was made. Among them were 7 cases with calcification at the lateral epicondyle; this represents 35 per cent. A similar study of painful shoulders during the same period of time was carried out for comparison. Cases in which a diagnosis other than periarthritis calcarea could be made either roentgenographically or by other methods were excluded from these statistics. Positive clinical diagnosis was made in 146 cases, 55 of these (38 per cent) showing evidence of calcification.

Fig. 3. Roentgenogram of an anatomic specimen. Usual appearance of the distal end of the humerus. This roentgenogram was taken for comparison with Figures 4, 5 and 6, as part of a roentgen-anatomic study of the elbow.

In another statistical study we reviewed the relative incidence of calcification at the lateral and medial epicondyle of the humerus as discovered in roentgenograms and compared with figures of painful elbows (tennis elbow) regarding the relative involvement of the lateral and medial aspects of the elbow—as first observed on clinical examination. On the basis of our concept a parallelism in both diseases was expected and found. During the two-year period in which calcification at the lateral epicondyle was discovered in 7 cases, 1 patient had calcification at the medial epicondyle.[10] This (7:1) ratio of calcification at the lateral and medial epicondyles is almost identical with that of clinically observed cases of epicondylitis (43:6) reported by Spencer and Herndon.[22]

On the basis of these comparative studies

Fig. 4. Roentgenogram of an anatomic specimen; distal end of a (right?) humerus. A faint bony shadow is seen at the lateral epicondyle due to a prominent ridge. This is a variation of the normal anatomy.

it may be stated that tennis elbow and periarthritis calcarea display similar behavior respective to incidence of visible calcification about the elbow.

We have never seen roentgenographic evidence of periostitis or periosteal new bone formation of other nature at the lateral epicondyle connected with this syndrome—as suggested by Cryiax,[7] Spencer and Herndon,[22] Maida,[15] and others. Periostitis is considered to be the result of a functional trauma of the tendons of the extensor muscles. A review of the available literature shows that this suggestion has not been substantiated by surgical or pathologic observations. The respective roentgen reports are vague and inconclusive. An anatomic and roentgen study performed by the author on anatomic specimens of 95 humeri of the George Washington University Medical School seems to clarify the previous misconception. In 9 instances, the ridge at the lateral epicondyle, serving for insertion of the common extensor tendon, was prominent to such a degree that on roentgenograms it cast a bony shadow of faint density. Similar observation was made at the medial epicondyle in 5 cases (Fig. 3, 4, 5 and 6). This is considered a variation of the normal, but can easily be confused with soft tissue calcification or periosteal new bone formation. Two such cases are demonstrated (Fig. 7 and 8). Figure 8 is a roentgenogram of a laborer who had the typical syndrome of tennis elbow on the left side. The density at the lateral and medial epicondyles was at first interpreted as calcium salt deposition in the adjacent soft tissues. Roentgenograms of the opposite side taken for comparison showed similar "changes." For a four month period, no change in the roentgenographic findings was observed. This is the case which has raised our interest in this problem and resulted in the roentgen anatomic study described above. After this study, the roentgenographic findings were re-evaluated and were considered to represent a variation of the normal anatomy.

The present study represents a new approach to the long debated questions of tennis elbow. This approach is not primarily an anatomic one, and it is intentionally so.

Fig. 5. Roentgenogram of an anatomic specimen; distal end of a (left?) humerus. Faint bony shadow is at the medial epicondyle. Variation of the normal anatomy.

The list of investigations based on pathoanatomic studies is relatively long and we selected to discuss in some detail two such reports which seem to support our concept of tennis elbow. The greatly divergent conclusions of the—individually valuable—reports of the literature regarding the pathologic substrate of tennis elbow, however, confuse rather than clarify the issue. It is believed that the anatomic approach is not very promising. The present study, as an attempt to bring the tennis elbow and periarthritis calcarea under a common denomination, is the result of such consideration. We believe that the two names encompass identical ideas merely expressed in different ways. Tennis elbow (golf elbow, epicondylitis, epicondylalgia) is a purely clinical term, whereas the concept of bursitis, tendinitis, peritendinitis and periarthritis calcarea has developed mainly from roentgenographic and pathologic observations. The conclusion of this study does not solve the question of etiology and pathogenesis of tennis elbow; the recognition, however, that this condition is identical with periarthritis calcarea opens new avenues to the problem, and by so doing, we feel, a step has been taken in the right direction.

Fig. 6. Roentgenogram of an anatomic specimen; distal end of a (right?) humerus. A faint bony shadow is seen at each epicondyle. Variation of the normal anatomy.

Fig. 7. Roentgenogram of the right elbow of a patient having symptoms of tennis elbow. Faint bony shadow is present adjacent to the lateral epicondyle. Compare with Figure 4. This is a variation of the normal anatomy. There is no visible calcium salt deposition in the soft tissues.

SUMMARY

A study of 20 cases of tennis elbow, observed during a two-year period in the Dispensary of the United States Army, The Pentagon, is presented. During this study it became evident that tennis elbow clinically and roentgenologically cannot be distinguished from periarthritis calcarea of the elbow. In 7 out of 20 cases of tennis elbow, calcium salt deposition adjacent to the lateral epicondyle of the humerus was discovered on roentgenographic examination. This was compared with the incidence of calcification in painful shoulders during the same period of time. The percentage of incidence was found very close (35:38). In another statistical study, the relative incidence of calcification at the lateral and medial epicondyles (discovered by roentgenographic examination) was compared

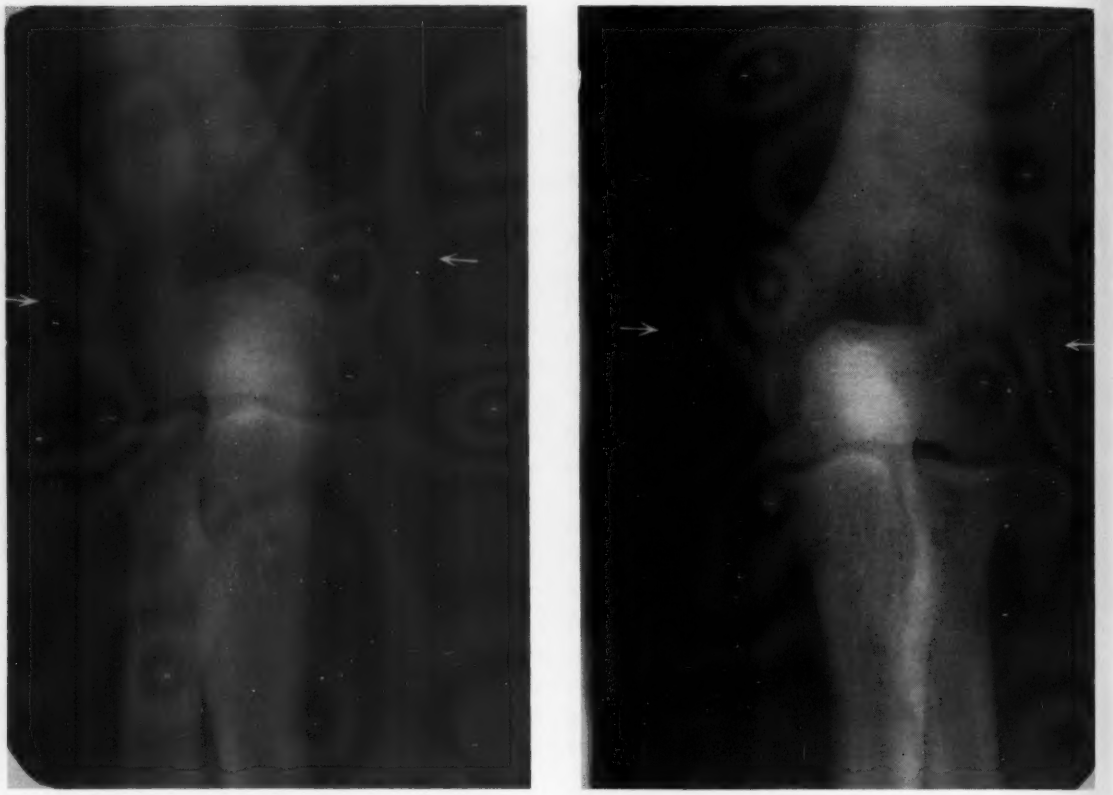

Fig. 8. Faint "calcific" shadow at the lateral and medial epicondyles of the humeri of a laborer who had symptoms of tennis elbow on the left. The "calcific" shadows are considered a variation of the normal anatomy.

with figures of clinical involvement on the lateral and medial aspects in tennis elbow. The relative incidence was almost identical (7:1) in both conditions. A roentgen study of anatomic specimens of humeri revealed that the appearance of a faint bony shadow at the lateral epicondyle is a variation of the normal; this had been in the past misinterpreted as calcium salt deposition or periosteal new bone formation and had been considered to be the underlying cause of tennis elbow. It is concluded that the tennis elbow syndrome is not a separate entity but that it represents a manifestation of periarthritis calcarea at the lateral epicondyle of the humerus.

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FRACTURES OF THE ELBOW JOINT

NEW CLASSIFICATION AND ROENTGENOLOGIC GUIDE TO MAJOR PITFALLS OF DIAGNOSIS AND TREATMENT

By JACOB KULOWSKI, M.D.
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PHYSICIANS who undertake the diagnosis and treatment of fractures of the elbow joint are generally aware of the clinical possibility of Volkmann's ischemic paralysis and/or contracture. However, alertness and anticipation in this regard based upon probabilities alone is not enough to avert such grave complications. In point of fact, the precise biomechanical requirements of function, the variable types of specific fractures, and the apparent urgency for reduction and immobilization of these injuries often serve to subordinate or mask these major pitfalls. The reversible stage of ischemic paralysis may be missed. Ultimately, the catastrophic (practically irreversible) stage of ischemic contracture supervenes and the opportune time for prophylaxis has passed.

Obviously, the situation calls for both improved diagnosis and treatment. One deterrent to this goal has been the lack of a more precise and prognostic classification of fractures involving the elbow joint. It is my purpose to provide such a classification, based upon critical anatomic factors which govern the clinical results and which may be approximately evaluated on roentgenologic evidence. These factors concern the joint capsule and the so-called vaginal (deep or ensheathing) fascia, and their relationships to the neurovascular bundle (brachial artery and median nerve) under the various conditions of fracture.

ROLE OF THE JOINT CAPSULE

Figures 1 and 2 illustrate the approximate confines of the joint capsule and its relationships to the neurovascular bundle (brachial artery and median nerve), radial nerve, ulnar nerve, and the vaginal (deep or ensheathing) fascia. The capsular compartment contains only the articular portions of the humerus, radius and ulna and the synovial membrane. All other soft structures of importance to our discussion are outside of the capsule. It becomes apparent that the precise—albeit limited—areas of capsular division at the elbow joint suggest a broad categorization of fractures which might meet the biomechanical requirements of clinicians everywhere and in the various practices they pursue, specialized or otherwise.

Table 1 indicates that these fractures lend themselves properly to classification according to intracapsular, extracapsular, and combined intracapsular and extracapsular groupings. The clinical implications with regard to intracapsular fractures are quite clear cut. First, it may be accepted that, with rare exceptions, intracapsular frac-

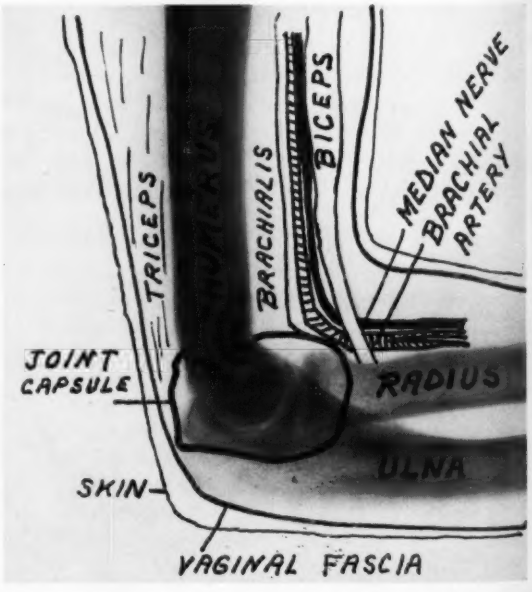

FIG. 1. Approximate interrelationships of the soft parts about the elbow joint. Particular attention is called to the normally protected position of the neurovascular bundle in the groove between the biceps and brachialis muscles.

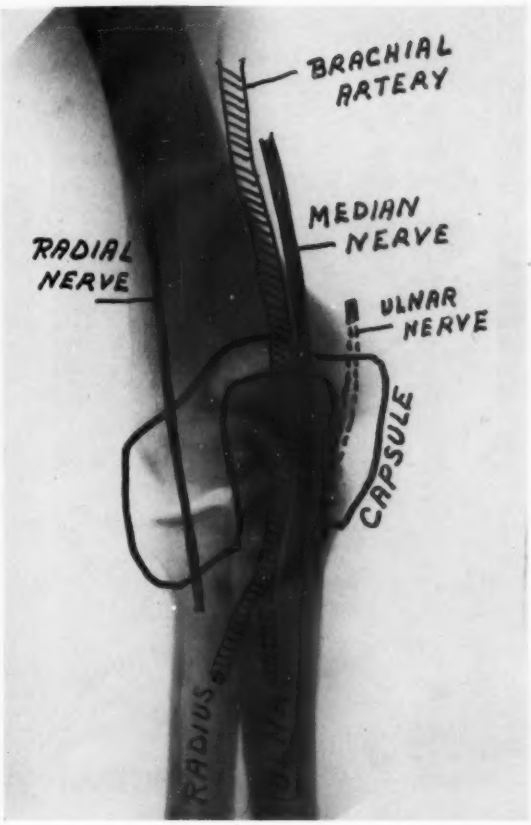

FIG. 2. Soft tissue approximations in the antecubital area, front view.

tures—with the capsule intact—hold no threat to the neurovascular bundle. In other words, intracapsular fractures are not likely to be complicated by ischemic paralysis, contracture, or myositis ossificans. An exception to the rule involves dislocations (Fig. 3) in which varying degrees of capsular tearing occur. Occasionally, in these cases there result some causalgic symptoms due to stretching of the neurovascular bundle over the smooth rounded

FIG. 3. Stretching action of the lower end of the humerus upon the neurovascular bundle in a case of posterior dislocation of the radius and ulna.

TABLE I

PILOT BIOMECHANICAL CLASSIFICATION OF ELBOW FRACTURES

Type	Humerus	Radius	Ulna
Extracapsular	Supracondylar Medial epicondyle	Bicipital tuberosity	"Monteggia"
Intracapsular	Trochlea Lateral condyle Capitellum Medial condyle	Head Neck "Monteggia"	Coronoid process
Combined (both extracapsular and intracapsular)	Dicondylar Y or T Supracondylar "Sideswipe" "Gunshot"	"Sideswipe" "Gunshot"	Olecranon "Sideswipe" "Gunshot"

Note: In addition to the above fractures, it is to be remembered that all dislocations involve the capsular areas. Common combinations of fracture dislocation involve the coronoid, medial epicondyle, head of radius, olecranon, lateral condyle of humerus and the Monteggia fracture dislocation.

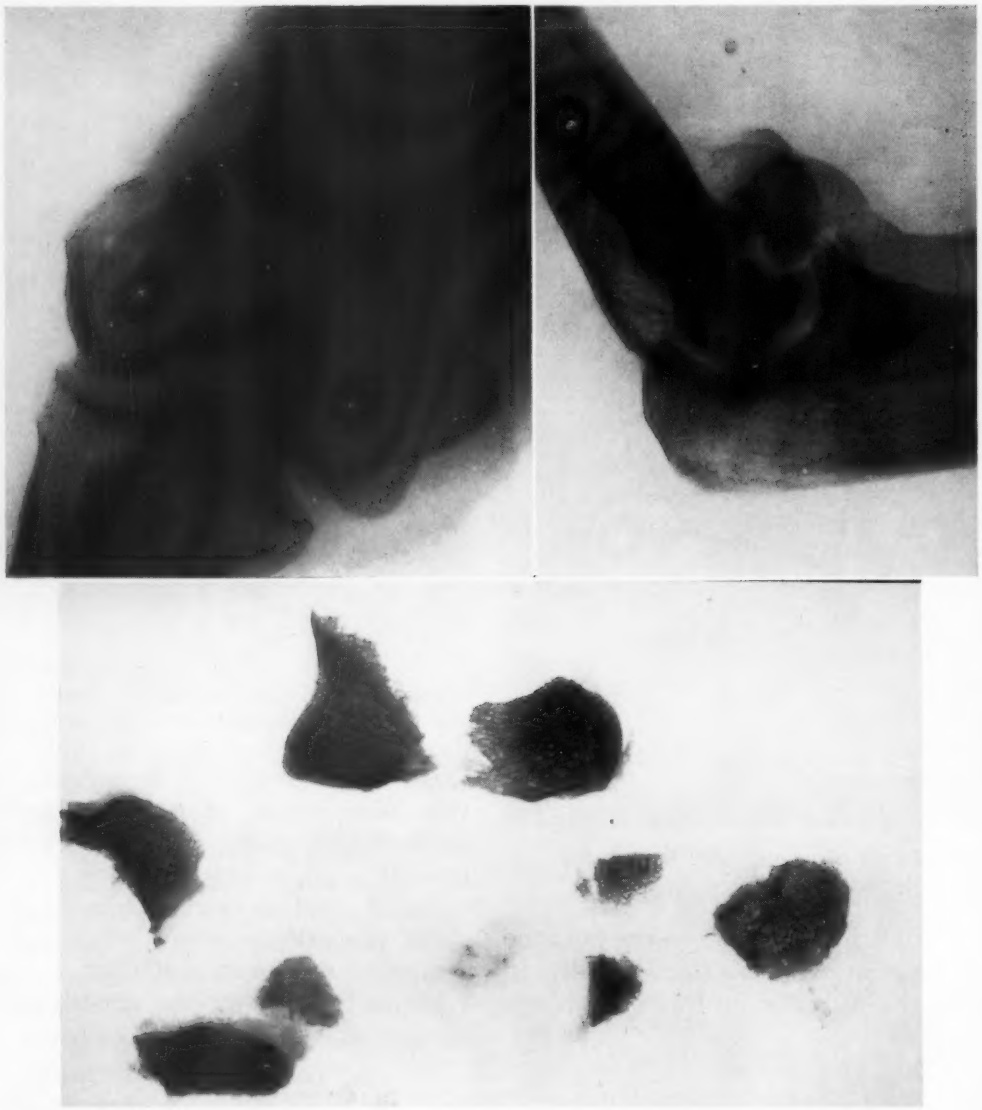

Fig. 4 One reason why intracapsular fractures threaten the future integrity of the joint, *i.e.*, the occasional discrepancy between the roentgenologic and operative findings. Many precise views are sometimes necessary to show the actual injury to the joint surfaces.

lower end of the humerus (in posterior dislocation of the radius and ulna).

On the other hand, intracapsular fractures demand very accurate reduction and fixation until adequate healing has taken place in order to avoid subsequent posttraumatic arthritis, contractures of the joint capsule and synovial membrane, formation of joint mice and other bony blocks to normal joint movements (Fig. 4). Intracapsular fractures are therefore not benign from the standpoint of ultimate anatomic and physiologic results. For these reasons operative treatment is frequently indicated in these cases.

Role of the Vaginal or Deep Fascia

Referring to Figures 1 and 2, it is noted that all muscles, nerves, and blood vessels (with the exception of the superficial veins

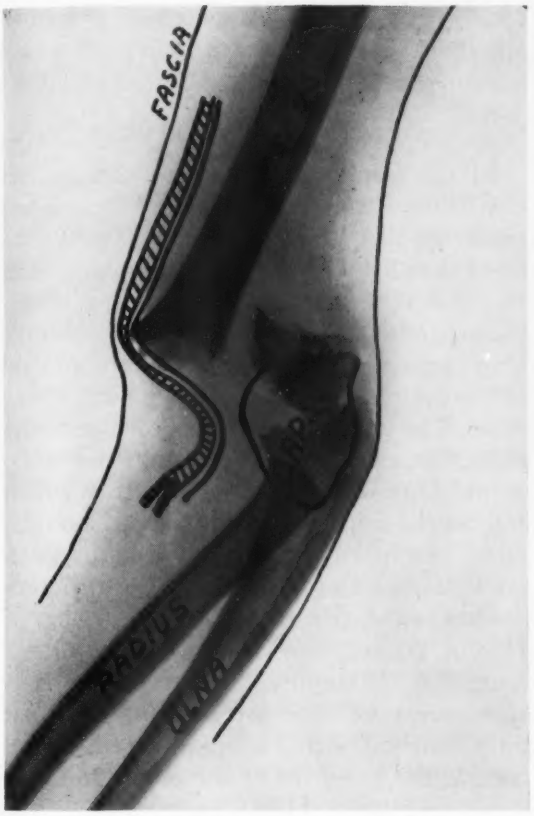

Fig. 5. This kind of impingement of the neurovascular bundle upon the lower end of the humeral fragment definitely threatens the immediate or early integrity of the lower arm and hand from ischemia. It is not infrequent in extracapsular supracondylar fractures, and may appear in varying degrees from any kind of extracapsular fracture. Moreover, the obvious laceration of the brachialis (note how close to the surface the lower humeral fragment is anteriorly) leads to subfascial tension from bleeding and edema.

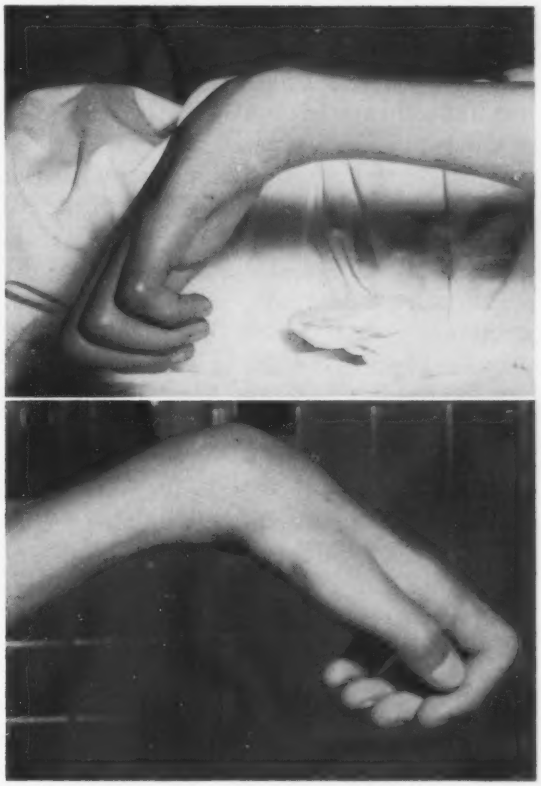

Fig. 6. The classic appearance of a Volkmann's ischemic paralysis and contracture. This was resistant to conservative methods of treatment, but yielded to operative lengthening of all flexor tendons.

which are on top of the fascia) are contained in the fascial compartment of the forearm, antecubital, and arm areas. It is apparent that any increased subfascial tension—from bleeding, edema or both, due to tight bandaging—squeezes the soft structures under the fascia because there is little or no yield to the fascia, the tensile strength of which has been determined to be like that of soft steel.[1] It is also evident that all extracapsular fractures tend to invade this fascial compartment by hemorrhage and edema of the soft parts. Moreover, certain of these fractures directly impinge upon the neurovascular bundle (Fig. 5), most commonly, of course, the supracondylar ones.

In addition to increased subfascial tension (bleeding, etc.) and direct impingement of the lower end of the fractured humerus upon the neurovascular bundle, there is the element of so-called "reflex arterial segmental spasm" of the brachial artery. I believe that the ultimate degree of ischemic complications depends upon the amount of participation by each or all of these mechanisms. In other words, the action of all three mechanisms, unchecked therapeutically, would most certainly result in a severe degree of paralysis and contracture of the forearm muscles and thus cripple the hand (Fig. 6). Severe subfascial tension (with or without the aggravating influence

of additional external pressure from splints or casts), unchecked therapeutically, may be expected to do likewise because of the mechanical interference with the collateral circulation of the brachial artery as well as the latter itself. Acting alone, perhaps the least damaging mechanism is that of the reflex segmental arterial spasm. This statement is based upon the assumption that spasm is secondary to direct bony impingement and/or increased subfascial tension pressure, and that both of these conditions are amenable to therapeutic controls.

ROENTGENOLOGIC IMPLICATIONS

The radiologist—the only ubiquitous consultant—can adequately evaluate the relationships of fractures to the joint capsule of the elbow. To clinch the diagnosis of a purely intra-articular fracture, he may request the doctor in charge to aspirate the distended joint, a procedure which gives added comfort to the patient, enhances closed reductive methods, and minimizes some post-traumatic complications.

In supracondylar fractures, the relative closeness of the anterior humeral fragment to the skin (Fig. 5) definitely indicates that the brachialis muscle has been lacerated, thus exposing the neurovascular bundle to both direct and indirect injury. Subfascial distentions will be clearly evident on roentgenograms, especially in cases in which the fascia has not been lacerated (as is the case in most simple fractures).

On the clinical side, impending ischemia is almost always heralded by pain, pallor or cyanosis, puffiness of the fingers and hand, and pulselessness at the wrist. Another important sign is the extreme pain resulting from passive attempts to extend such fingers. Occasionally, there is no pain. Moreover, the classic deformity of the wrist and fingers may be masked so long as the retentive apparatus maintains the wrist in the cock-up position. This becomes painfully apparent to the doctor as soon as the cast is removed.

CONCLUSIONS AND SUMMARY

Elbow fractures have been categorized into three main groups, *i.e.*, intracapsular, extracapsular, and combined types. The clinical and biomechanical implications of this grouping have been clarified. Uncomplicated intracapsular fractures involve long range results relative to anatomic and physiologic elements comprising the elbow joint. The critical factors concern accurate reduction or removal of irreducible fragments. On the other hand, all extracapsular fractures (especially the supracondylar ones) potentially threaten the neurovascular situation and, as such, require the immediate attention of the doctor in charge. Herein, consultation services are of urgent necessity. Juxtaposed intracapsular fractures endanger the integrity of the joint only whereas extracapsular fractures endanger the integrity of the entire forearm, wrist, and hand. Helpful suggestions from the radiologist to the physician in charge, with the aim of achieving the best clinical results are of greatest value.

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THE DIAGNOSTIC SIGNIFICANCE OF ROENTGENOLOGIC SOFT TISSUE SHADOWS IN THE PELVIS*

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ON A roentgenogram of good technical quality, certain soft tissue pelvic structures are plainly visible. A knowledge of the normal appearance of these structures and alterations which occur in disease is a valuable asset to diagnosis.

The difficulty of obtaining equally good visualization of denser areas, such as barium-filled segments of the colon and bone, as well as the soft tissue shadows especially in lateral views on a single film of standard exposure, is well known. For this reason, the soft tissue shadows should be carefully examined with a spot light. None of the accompanying illustrations shows the soft tissue shadows as well as they can be seen on the original films, but, we believe, they will serve a useful purpose in introducing, orienting and emphasizing certain important observations helpful in diagnosis.

On the plain roentgenogram made in the anteroposterior projection, one can usually see the bladder, the levator ani muscle, the obturator internus muscles, the sacrospinous ligaments, and, in the female, the uterus. The fibers of the gluteus maximus muscles are commonly seen traversing the pelvis obliquely. Not infrequently, the rectum and a knuckle of sigmoid are visualized by virtue of their gas or fecal content (Fig. 1 and 2).

In the female, the shadow of the bladder is distinctly separated from the uterus by a radiolucent "cleavage plane." The importance of this cleavage plane is readily appreciated when one is confronted with the problem of diagnosing a pelvic tumor (Fig 2 and 3). A diagnosis of pelvic tumor should not be made if the bladder cannot be identified unless there is pathognomonic evidence, such as the calcification which occurs in leiomyoma of the uterus or dermoid cyst of the ovary.

A cleavage plane also delimits the contour of the bladder in the male, or it may be visualized because of its contents. It has long been our practice to make lateral[1] as well as posteroanterior roentgenograms before and after evacuation of a barium

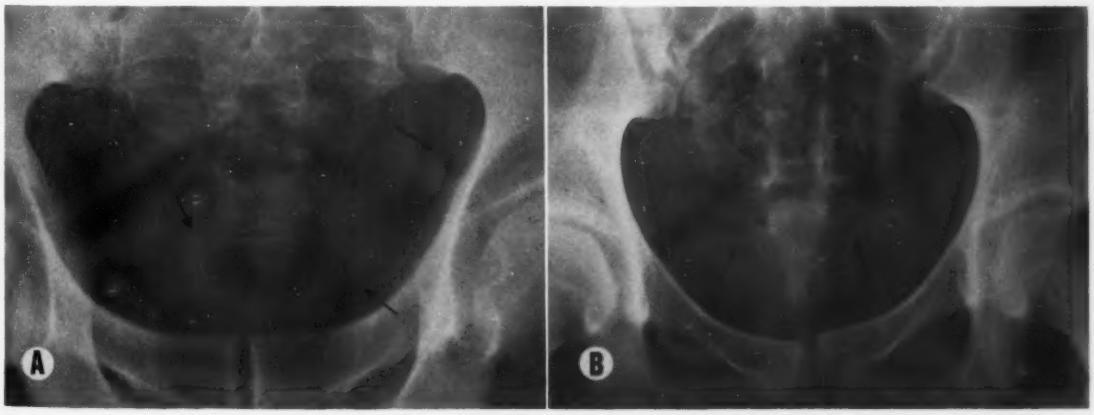

FIG. 1. (*A*) The obturator internus muscle (upper right arrow), levator ani muscle (lower right arrow) and bladder are visualized because of interposed fatty areolar tissue. (*B*) The sacrospinous ligaments. Normally, they are symmetrical and appear as curved linear densities, convex upward. They can be traced from the coccyx to the ischial spines.

* From the Departments of Radiology, Massachusetts Memorial Hospitals and Boston University School of Medicine.

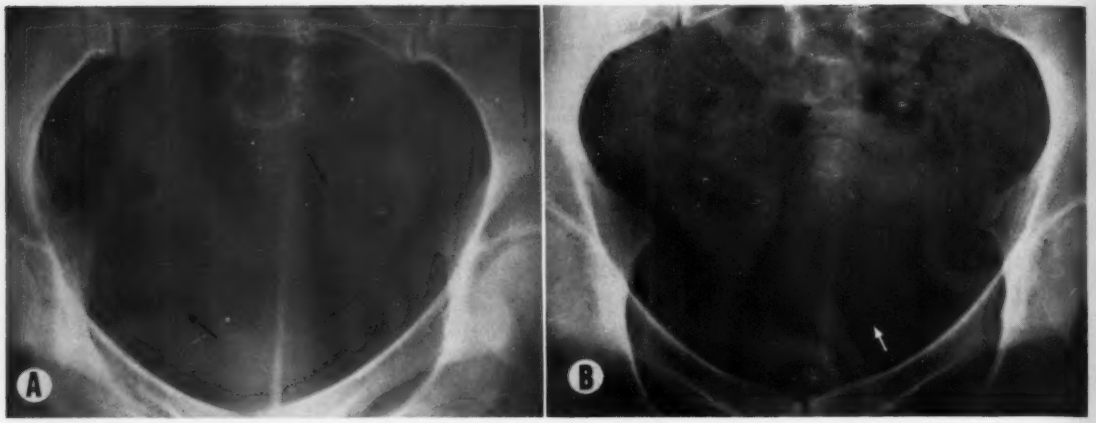

Fig. 2. (*A*) Fibers of the gluteus maximus muscles. The sigmoid is seen by virtue of its contents (upper arrow). (*B*) A "cleavage plane" separates the shadows of the bladder and uterus. The bladder must be identified before making a diagnosis of pelvic tumor (see Fig. 3).

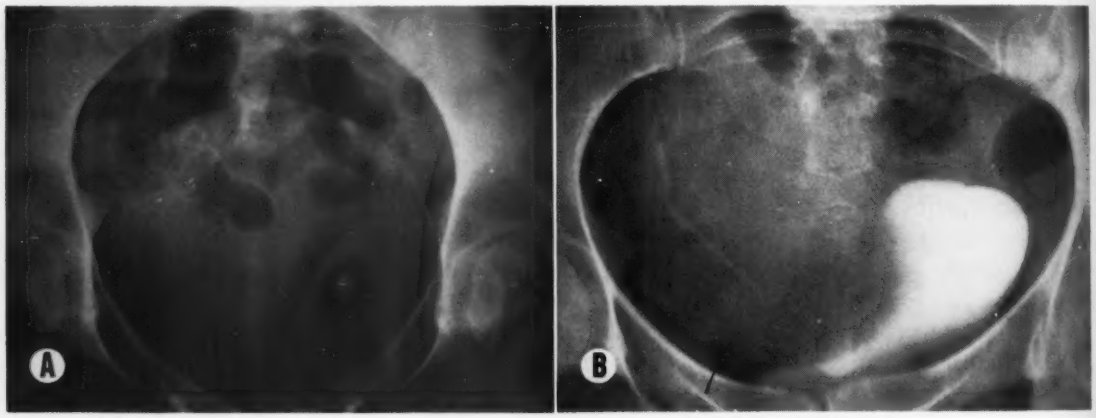

Fig. 3. (*A*) Distended bladder in a female. Note the absence of a cleavage plane (see Fig. 2*B*). A diagnosis of pelvic tumor should not be made until the bladder has been emptied by voiding or catheterization. (*B*) Pelvic tumor. Note the cleavage plane which defines the bladder. A diagnosis of pelvic tumor may be made with safety.

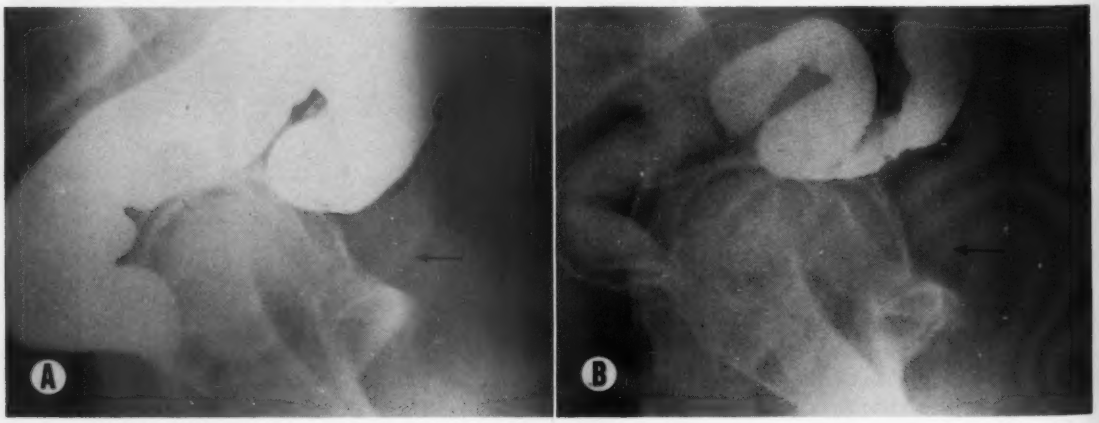

Fig. 4. (*A*) Routine lateral roentgenogram made during examination of the colon by barium enema. Note the distended bladder (arrow). (*B*) Routine lateral roentgenogram made after evacuation of the barium enema. Note that the bladder is still distended (arrow). This suggested that "such a state is not physiologic" and prompted a diagnosis of prostatism. (An enlarged prostate was found on rectal examination.)

Fig. 5. Routine lateral roentgenogram made during examination of the colon by barium enema. The normal bowel, when distended, is bilaterally convex. Note the area of concavity (arrow). A diagnosis of probable uterine fibroid was confirmed by other examinations. (Reprinted by courtesy of *Medical Clinics of North America.*)

enema in examination of the colon. Figure 4 is presented as an interesting finding deduced by study of the soft tissue shadows. It will be noted that the size of the bladder in the postevacuation film is as large as it was in the film showing the distended colon. It was our belief that "such a state is not physiologic" and that the probable cause was prostatism. Subsequent examination showed the patient to have an enlarged prostate. We have also found the lateral view to be extremely helpful in detecting pelvic tumors (Fig. 5).

The levator ani muscle presents a crescentic shadow of increased density on each side of the pubic symphysis with its convexity directed into the pelvis. Not infrequently, the levator ani produces a pressure deformity of the opacified bladder somewhat similar to that produced by an enlarged prostate. This appearance is also seen in women (Fig. 6). It will be noted, however, that the defect in the bladder outline produced by the levator ani muscle consists of two semilunar indentations which meet in the mid-line to form a pointed projection. In the case of bladder deformity due to an enlarged prostate, the central projection is usually absent. A single concave pressure defect which resembles an enlarged prostate, but which is also found in women, is due to an enlarged retropubic fat pad (Fig. 7).

Not infrequently, the shadows of the sacrospinous ligaments are sufficiently dense so that they appear to transect the opacified bladder and simulate the appearance of an enlarged prostate (Fig. 8). Differentiation is readily made by tracing the shadows of the sacrospinous ligaments from their origins on either side of the coccygeal and lower sacral segments to the ischial spines. They normally appear curved, with their convexity upward, and they taper to a point at their insertions in

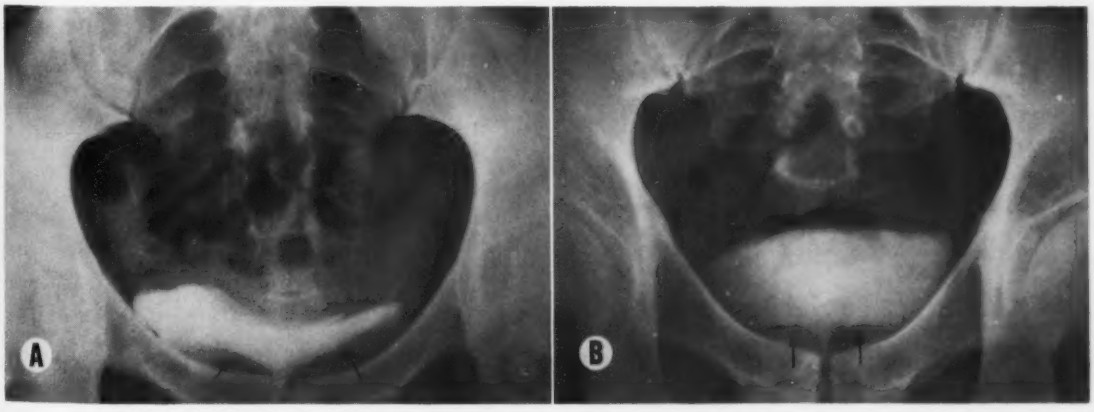

Fig. 6. (*A*) Pressure deformity on the base of the bladder produced by the levator ani muscle (arrows). Female patient. (*B*) Pressure deformity on the base of the bladder produced by the levator ani muscle (arrows). Male patient. Both roentgenograms were made with patients supine, *not* voiding. Note how the convexities meet to form a pointed projection. (Compare with Fig. 7.)

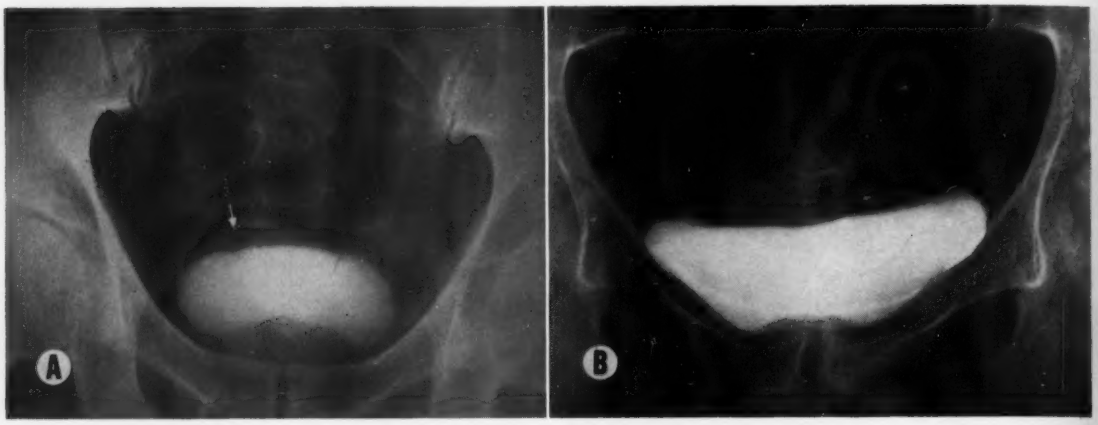

Fig. 7. (*A*) Typical deformity of the bladder produced by an enlarged prostate (compare with Fig. 5 and 8). Arrow points to thickened bladder wall. (*B*) Typical deformity of the bladder in female patient produced by an enlarged retropubic fat pad. This can, of course, also be seen in the male.

Fig. 8. (*A*) The sacrospinous ligaments (arrows) are dense and appear to transect the bladder. The appearance may be so pronounced as to simulate prostatic hypertrophy. Female patient. (*B*) The dense sacrospinous ligaments present an illusion of prostatic hypertrophy (left arrow). Upper right arrow points to obturator internus; lower right arrow points to levator ani. Male patient.

Fig. 9. Calcified sacrotuberous ligament (right arrow). Unlike the sacrospinous ligaments these are usually not visible unless calcified. Calcified sacrospinous ligament on left.

the ischial spines. The sacrospinous ligaments may be torn during difficult labor, especially if the application of forceps was necessary, and one of them is frequently deformed or displaced with fracture of the coccyx.

The sacrotuberous ligaments are much less commonly seen on a plain roentgenogram. Sometimes they are calcified and, in such cases, present a serious obstacle to the normal mechanism of parturition (Fig. 9 and 10).

The obturator internus muscles appear as two semilunar shadows of increased density on the inner aspect of the pelvis (Fig. 1 *A*). They overlie the pelvic surface

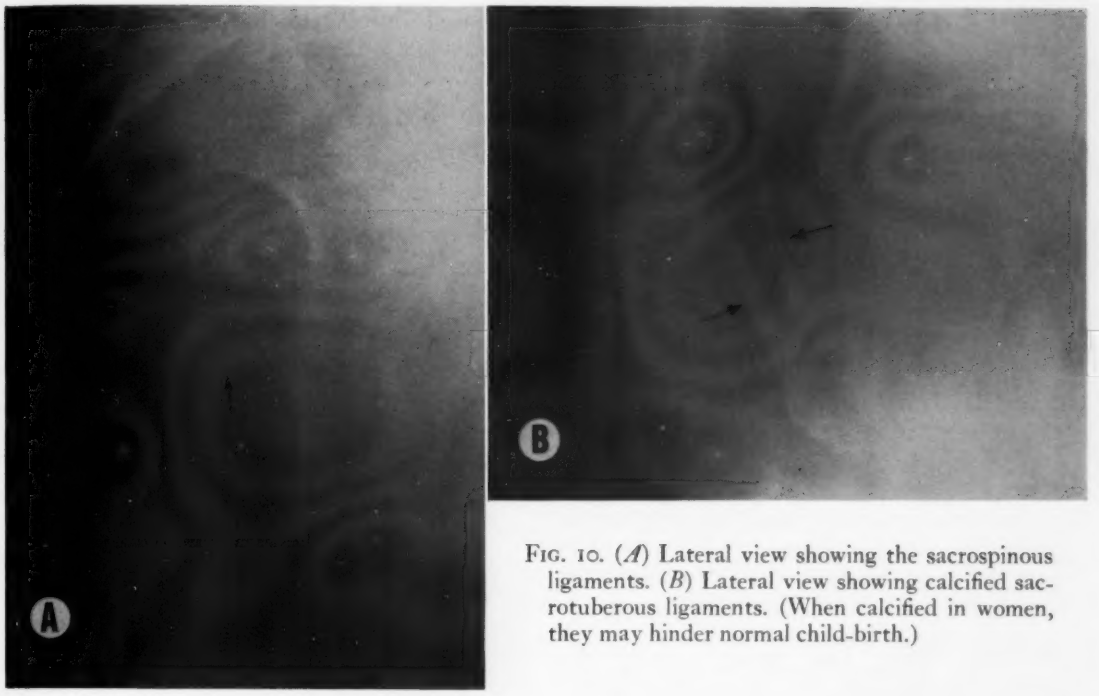

Fig. 10. (*A*) Lateral view showing the sacrospinous ligaments. (*B*) Lateral view showing calcified sacrotuberous ligaments. (When calcified in women, they may hinder normal child-birth.)

of the acetabula and extend posteriorly on the inner surface of the iliac bones. They may become prominent in any disease which affects the hip: infection, such as tuberculosis, fracture or tumor (Fig. 11). Increased prominence of the obturator internus on one side may appear earlier than the osteolytic lesions of metastatic disease. Thus, an elevated obturator internus muscle contributes confirmatory evidence in the evaluation of early structural changes in the bone in a question of metastasis.

The gluteus maximus muscle usually offers no problem in differential diagnosis. We have seen one case, however, in which the fibers of the gluteus maximus were unusually coarse. In a lateral view taken elsewhere during examination of the colon, the cross-section of these fibers showed sharp spiculation and was interpreted as "ray formation typical of osteogenic sarcoma of the sacrum" (Fig. 12).

On a plain roentgenogram made in the lateral position one can see the anterior

Fig. 11. (*A*) The obturator internus is markedly prominent due to a hematoma resulting from fracture at the right iliopectineal eminence (left arrow). (*B*) The obturator internus is prominent due to metastatic involvement of the left hip.

Fig. 12. "Ray formation," diagnosed elsewhere as osteogenic sarcoma of the sacrum. The spiculation is due to the normal gluteus maximus muscle, the fibers of which are seen in cross-section.

abdominal wall, the rectus abdominis muscle, the bladder, the symphysis pubis and the space of Retzius. In certain diagnostic problems much useful information may be obtained from a lateral view. The patient should be placed in a true lateral position with the thighs straight or extended. The natural tendency to flex the thighs to facilitate balance when lying on the side militates against best visualization of the important structures in question, since, in this position, the thighs may obscure the soft tissues about the pubis.

The anterior abdominal wall, the rectus abdominis muscle, the bladder and the space of Retzius are clearly defined by cleavage planes due to a variable amount of adipose tissue between these structures (Fig. 13). In cases of pelvic abscess with pelvic peritonitis the cleavage planes are obscured and the shadows of the anterior abdominal wall, rectus abdominis muscle and bladder may fuse into one homogeneous density. The same phenomenon is seen in the anteroposterior roentgenogram where the bladder, uterus and surrounding soft tis-

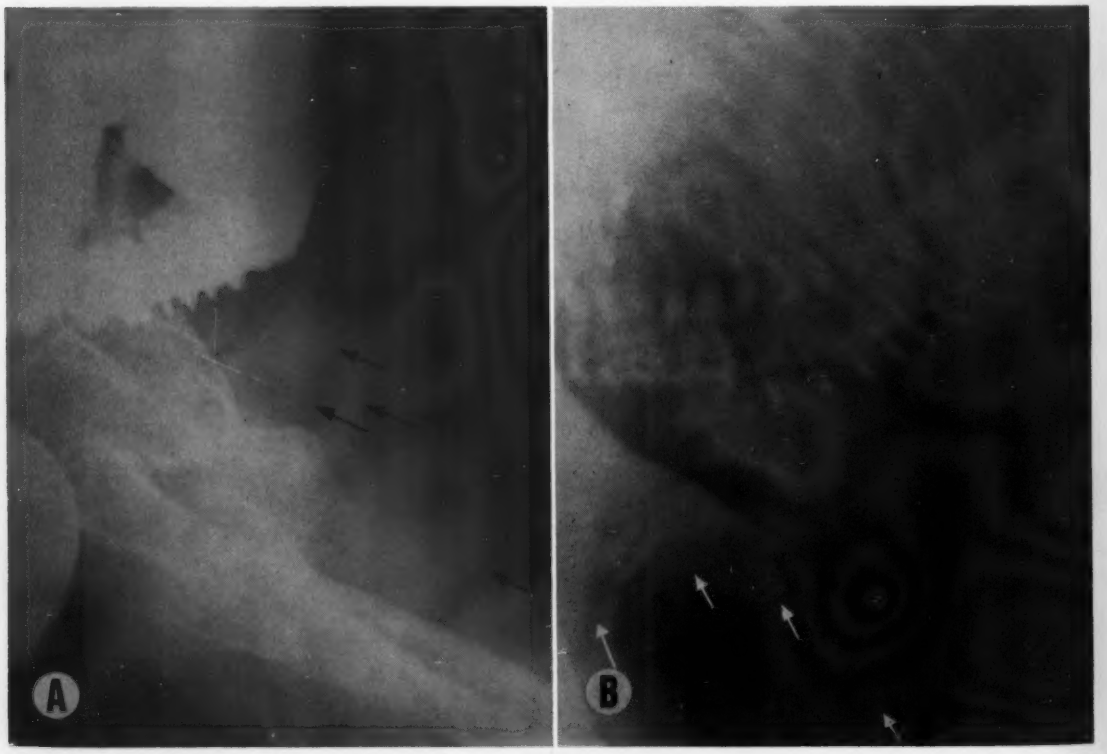

Fig. 13. (*A*) Routine lateral roentgenogram made during examination of the colon by barium enema. From above downward, the arrows point to: the distended bladder, rectus abdominis muscle, the space of Retzius and the anterior abdominal wall. (*B*) From left to right the arrows point to: the bladder, lower uterine segment, rectus abdominis muscle and anterior abdominal wall (compare with Fig. 15).

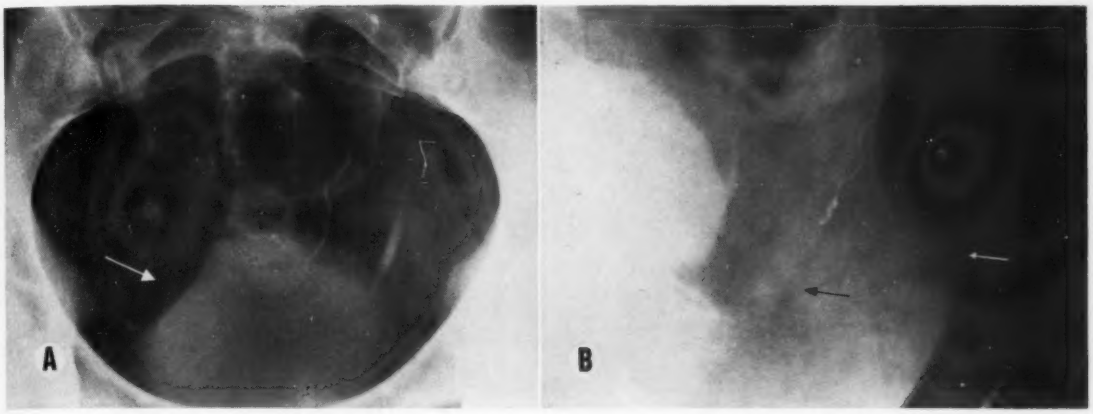

FIG. 14. (*A*) Extrinsic pressure deformity of the bladder due to a pelvic mass. (*B*) Note the complete loss of cleavage planes. White arrow indicates position of anterior abdominal wall. Black arrow shows the faint outline of the rectus abdominis muscle. Loss of cleavage planes is found in pelvic abscess or pelvic peritonitis. This patient had an abscess in the space of Retzius (compare with Fig. 13).

sues also appear fused and the cleavage planes are lost. The roentgenogram usually shows mottled gas shadows, but the distribution and contour of these shadows do not correspond to a normal anatomic segment, from which it may be deduced that they are not contained within a segment of intestine or rectum. Thus, they offer a clue to the possibility of pelvic abscess or peritonitis (Fig. 14 and 15). It must be emphasized that in making such examinations the bladder should be emptied by voiding or catheterization, since a distended bladder may rise out of the true pelvis and obscure surrounding soft tissue structures (Fig. 3). Ascitic fluid in the pelvis may also obscure structural outlines. This problem is usually resolved by taking an anteroposterior roentgenogram in the Trendelenburg position.

Study of this region in the lateral position is helpful also in determining the question of extrauterine pregnancy. In the normal pregnant state the lower uterine segment is visualized and its borders extend into the abdomen to encircle the fetus.[2] In cases of ectopic or abdominal pregnancy after the first trimester, the fetus may *appear* to be contained within the uterus, but careful study will reveal that the containing sac does not extend into the pelvis behind the bladder to cor-

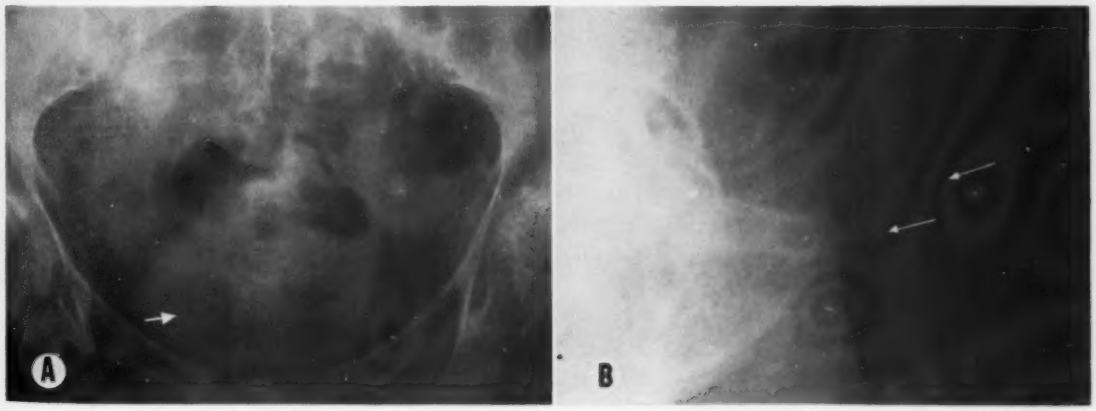

FIG. 15. (*A*) Pelvic abscess. Note abscence of cleavage planes and the mottled gas shadows (arrow). The distribution of these gas shadows does not conform to the anatomic contour of the sigmoid or rectum. (*B*) Lateral view. The cleavage planes are almost entirely obliterated. This appearance is typical of the pelvic inflammatory disease. (Reprinted by courtesy of *Medical Clinics of North America*.)

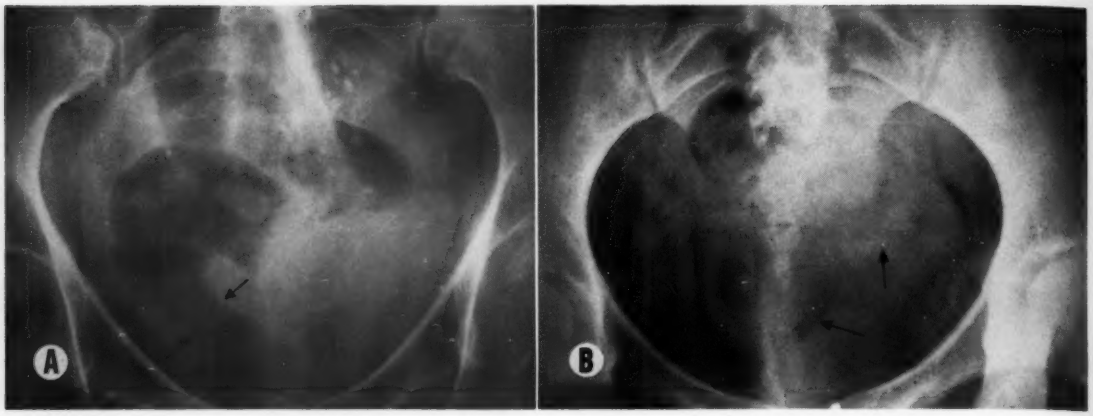

Fig. 17. (*A*) Deformed contour of gas-containing rectum and sigmoid strongly suspicious of carcinoma of the rectum (confirmed). (*B*) Thin crescentic shadow (lowest arrow) indicating compression and displacement of the rectum, arousing suspicion of pelvic tumor (confirmed). The left sacrospinous ligament is depressed (right arrow). The bladder is distended. No cleavage plane is visible. Left arrow points to right sacrospinous ligament (compare with Fig. 3).

respond to the normal appearance of the lower uterine segment (Fig. 16; compare with Figure 13).

Not uncommonly a circular area of density 2 or 3 cm. in diameter may be seen in the left side of the pelvis, and, on occasion, this has been mistaken for a tumor. It is produced by a knuckle of sigmoid rendered visible by its fecal content. In women, it usually appears in the region of the left ovary (Fig. 2 *A*).

Fig. 16. Extrauterine pregnancy. Arrows point to the rectus abdominis muscle, anterior abdominal wall and pubic symphysis. Note the absence of the shadow of the lower uterine segment (see Fig. 13 *B*). (Reprinted by courtesy of "*Medical Clinics of North America.*)

It is helpful also to acquire familiarity with the rectum when visualized by its gas content. When seen in any position, the normal outline of the distended rectum is bilaterally convex. Deformity of the gas shadow may be the first clue to the presence of an organic lesion of the rectum or a pelvic mass (Fig. 17).

SUMMARY

Familiarity with the appearance of soft tissue shadows in the pelvis in the normal state and in disease is a helpful adjunct to diagnosis. Illustrations are presented to show the normal appearance and pathologic variations.

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A ROENTGEN METHOD OF EVALUATING DENSITY OF BONE*

By STANLEY B. REICH, M.D., JOSEPH LEVITIN, M.D., *and* LELAND R. FELTON, M.D.
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DECALCIFICATION of bone may be the result of osteoporosis or osteomalacia.

Osteoporosis is a condition characterized by a deficiency in the organic matrix of bone and usually is the result of lack of activity of the osteoblasts. This deficiency of matrix causes a deficiency of minerals usually bound to the proteins. It is found in many conditions such as disuse, menopause, senile states and endocrine derangements.

Osteomalacia is a condition in which there is a defect of calcification of bone matrix. This can be found in any condition causing deficient calcium absorption from the gastrointestinal tract or excessive calcium excretion from the body.[3]

These conditions are characterized by loss of "bone density" on roentgenograms and we shall use the term demineralization to include both.

Babaiantz[1] and others have found that after 30 per cent decalcification of nonmacerated bone demineralization can be suspected roentgenographically. However, a loss of 50 per cent was necessary for the roentgen findings to be unequivocal.[1] Therefore, many cases of borderline demineralization are missed and it is extremely difficult

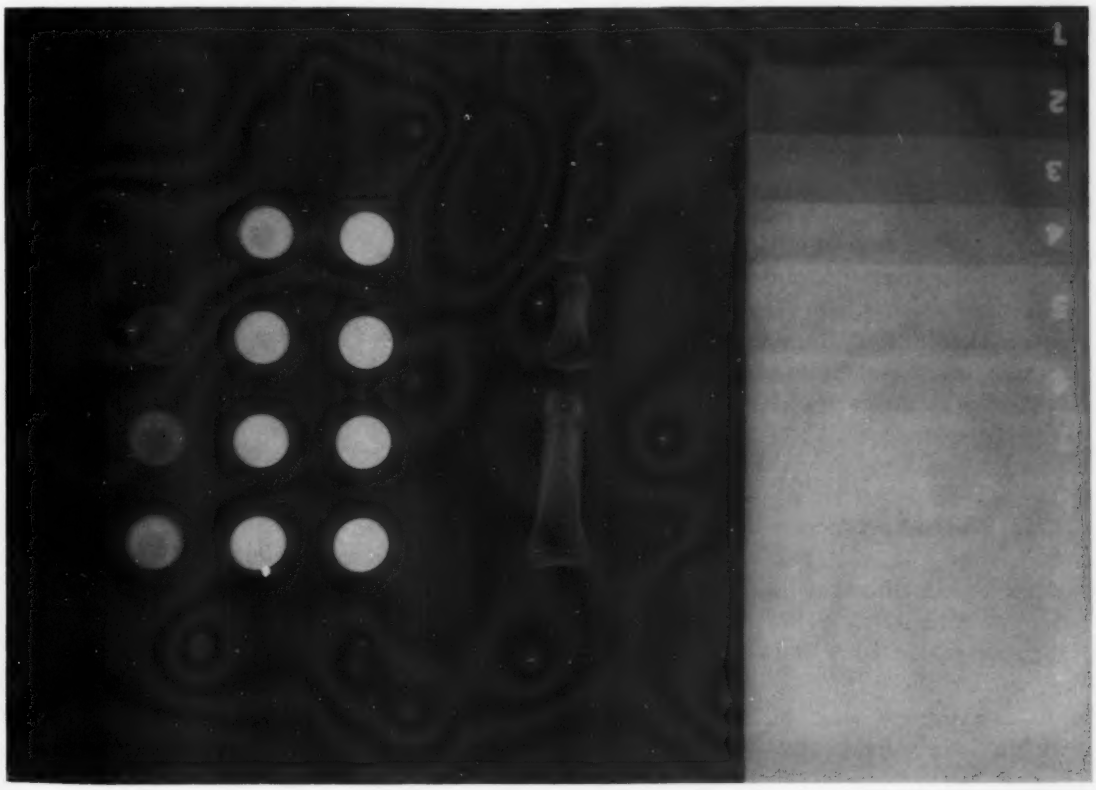

FIG. 1.

* From the X-Ray Department, Mount Zion Hospital, San Francisco, California. Presented before the IV Congresso Medico Dos Radiologistas e Electrologistas de Cultura Latina, Lisbon, Portugal, April, 1957.

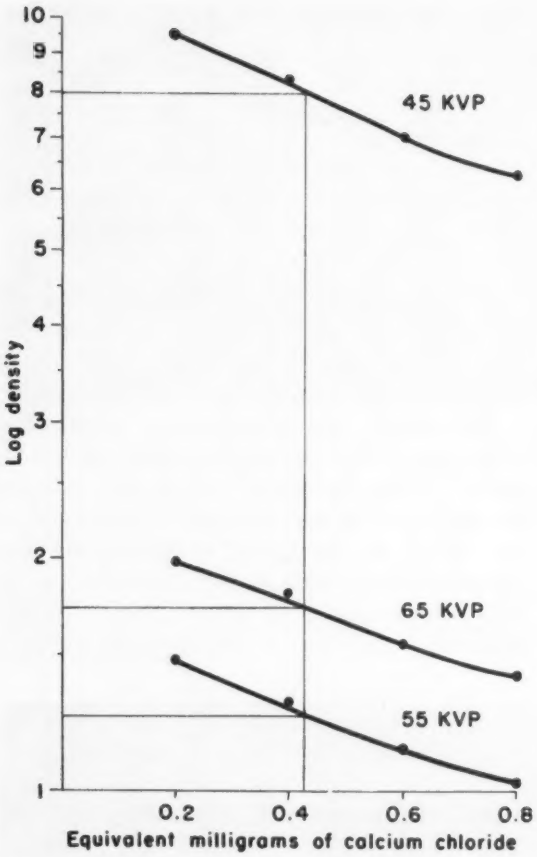

Fig. 2. Effect of variation of kilovoltage demonstrated with the calcium chloride "wedge."

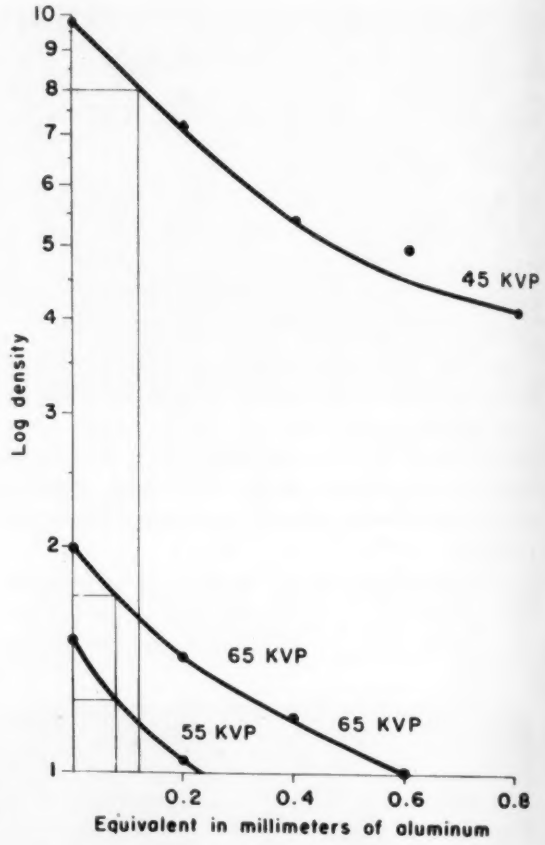

Fig. 3. Effect of variation of kilovoltage demonstrated with the aluminum "wedge."

to follow the effect of treatment on obvious demineralization.

Many attempts have been made to measure calcification of bone by the use of step wedges and fixed exposure-development techniques.[2,4,5,7–11]

METHOD

Our method consists of densitometric analysis of the calcification of the phalanges. This is done by making a roentgenogram of the hand submerged in a fixed depth of water in a machined aluminum pan with a one millimeter base. An aluminum step wedge and a calcium chloride "wedge" are submerged in the same pan (Fig. 1).

The calcium chloride "wedge" was made by drilling round one centimeter diameter holes in a one centimeter deep lucite block. These were filled with graded solutions of calcium chloride and each surface was closed with cover glasses.

The simultaneous exposure of wedge and bone eliminates much of the difference in development, emulsion, and exposure inherent in any two film method.

The submersion of the part and wedges in water minimizes the correction necessary for soft tissue density. The only difference in absorption is caused by the slight relative radiolucency of fat. Under the conditions of this experiment the opacity in a finger with soft tissues is approximately 2 per cent less than the isolated bone.

The opacity of the film was measured by a Kodak color densitometer of the diffusion type. Each reading covered an area $1\frac{1}{4}$ mm.

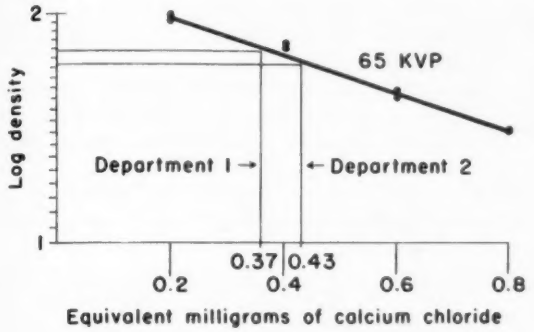

FIG. 4. Result of changing roentgenographic and dark room techniques (different radiologic departments).

in diameter. Non-screen film was used because it registers a greater range of density than screen film.

We chose the lateral proximal medullary portion of the second phalanx of the index finger as a standard. This area was easily reproduced in a film and represents two relatively thin cortices and medullary bone superimposed. It is also possible to get the anteroposterior diameter of this part relatively accurately on lateral films.

The opacities of the calcium standard are plotted on a graph. The aluminum wedge is plotted as a check. The density of the bone is plotted against the curves of standard. The equivalent density in milligrams of calcium chloride in solution (or millimeters of aluminum) is obtained (Fig. 2 and 3). It is possible to refine this to equivalent milligrams of calcium per cubic millimeter of bone scanned.

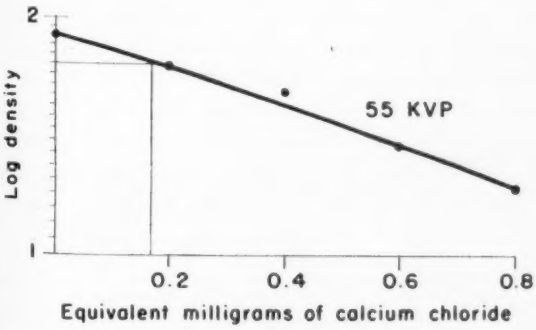

FIG. 5. First analysis of the demineralized bone.

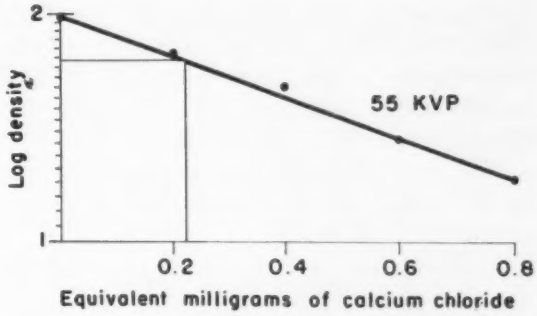

FIG. 6. Second analysis of the demineralized bone.

The effect of kilovoltage was evaluated by taking roentgenograms of phalangeal bones submerged in the apparatus and altering the kilovoltage. The variation was less than 2 per cent over a 20 kv. range (Fig. 2 and 3).

The same setup was moved to another radiologic department and similar films obtained. The variation with change of roentgen ray machine, exposure, and development was approximately 14 per cent (Fig. 4).

Subsequently the bones of the phalanges were split. One-half was analyzed for milligrams of calcium per cubic millimeter of bone. The other half was partially demineralized and roentgenographed again. Densitometric estimates of its mineralization were obtained (Fig. 5 and 6).

The partially demineralized bones were then chemically analyzed for milligrams of calcium per cubic millimeter of bone.

The results of the roentgenographic estimate and chemical analysis are shown in Table I. These indicate that the method of

TABLE I

DEMINERALIZATION OF PHALANX

Roentgenographic Estimate	
Equivalent milligrams of calcium chloride	
Average value before demineralization	0.40
Average value after demineralization	0.20
Chemical Analysis	
Calcium in milligrams per cubic centimeter	
Before demineralization	0.31
After demineralization	0.11

roentgenographic estimation of calcification is capable of picking up changes of approximately 15 per cent. This is a marked improvement over the usual roentgenographic error of 50 per cent.

The method outlined is primarily useful in following individual cases during the course of the illness.

CONCLUSION

A roentgenographic-densitometric method of estimating calcification of phalangeal bones is presented. It is capable of picking up changes of mineralization of approximately 15 per cent.

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Our thanks to Alvin Lewis, M.D. for the analyses of the specimens.

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A SOLID-STATE AMPLIFYING FLUOROSCOPE SCREEN*

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THE problems associated with the use of the conventional fluoroscope screen are well known to those who daily toil in darkness attempting to extract picture information from it. For those who have not used it, the Carman Lecture presented by Dr. W. E. Chamberlain in 1941 provides a convincing array of technical reasons why the directly viewed fluoroscope screen is an instrument of limited utility, causing discomfort to the observer and imposing a limit on exposure time because of the roentgen-ray levels required.

During the past decade a number of electron tube devices have been developed for reducing the roentgen-ray dosage and increasing the image brightness.[1,3,6,8] These have successfully demonstrated that a reduction in roentgen-ray dosage of about 10 times[10] is possible without loss of information content from the fluoroscope screen, and that dark adaptation can be eliminated by the many orders of magnitude brightness increase. However, the complexity, bulk, expense and operating problems associated in varying degrees with these systems have until now imposed limitations on their widespread use.

The objectives of the present work are to increase the image brightness and reduce the roentgen-ray dosage by using solid-state techniques. This approach allows the retention of many of the desirable features of the conventional fluoroscope screen since it permits the construction of the entire amplifying system in the form of a thin, large-area panel which can be easily viewed and manipulated, and which is free of complex auxiliary equipment.

The work to be described stems from our earlier work on solid-state light amplifiers.[4,5] With such panels it was demonstrated that a very dim image of visible light could be intensified by more than 100 times. The success in obtaining large gains in radiant energy with panels comparable in size to fluoroscope screens was an encouragement to extend this work into the field of fluoroscopy.

PRINCIPLE OF OPERATION

The basic principle of the amplifier is shown in Figure 1 which represents a single picture element. A photoconductor and an electroluminescent phosphor are connected electrically in series, and a-c voltage is applied across them. Photoconductive materials have the property of increasing their conductivity when excited by radiation. When unirradiated, many of these materials act as insulators. When excited with low levels of radiation, they increase their conductivity by many orders of magnitude. Depending on the particular material, they are sensitive to different types of radiation such as visible light, infrared, or roentgen rays in varying degrees. Electroluminescent phosphors are materials which emit light when voltage is directly applied to them, with the color of the light depending on the composition of the phosphor.

In the combination, the photoconductor acts as a valve or variable resistor controlling the flow of electrical power into the phosphor element. With sensitive photoconductors a low-level roentgen-ray beam can control a high level of output light. By comparison, in the case of the conventional fluoroscope phosphor, the output light is relatively low, deriving its energy from the conversion of the low-level energy in the roentgen-ray beam. Using the amplifying principle a picture device can be made with the materials in the form of thin layers. In Figure 2 the cross section of such an ampli-

* Presented at the Fifty-eighth Annual Meeting of the American Roentgen Ray Society, Washington, D.C., October 1–4, 1957. A scientific exhibit on the same subject was awarded the Silver Medal.

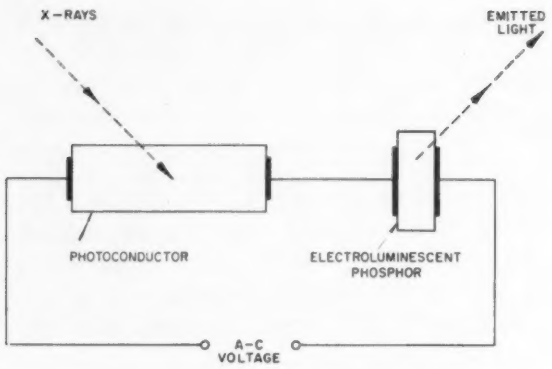

Fig. 1. Single-element amplifier.

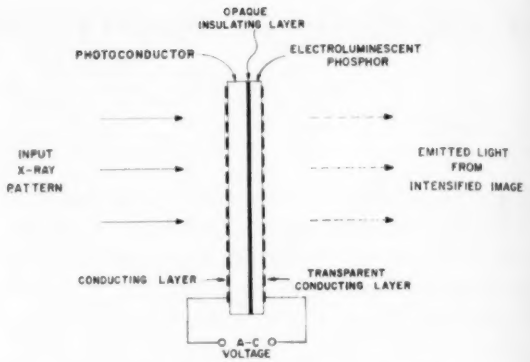

Fig. 2. Double-layer image intensifier.

fier is shown in idealized form. The two layers are placed in direct contact with an opaque insulating layer between them to prevent output light from feeding back to the photoconductor. Electrodes are provided on the outer surfaces, the electrode on the viewing side being transparent to visible light. Since the layers are very thin, roentgen rays exciting a local area of the photoconductor cause a flow of a-c current which is restricted to the adjacent phosphor area. This allows a pattern of roentgen rays to be converted to a pattern of light.

AMPLIFIER DESIGN

Because of the special properties of the photoconductive and electroluminescent materials used, the actual panel design differs from the idealized double-layer amplifier. Before describing the panel design, some of the properties of the materials will be discussed.

The phosphor is a Zn(S:Se) copper-activated material[2] emitting a yellow-green light. Typical of most electroluminescent phosphors, it is in powder form bonded with plastic. For operation such phosphor layers require a-c voltage to produce light. With d-c there is only a negligible current flow and no light emission. Operation is possible over a wide range of audio frequencies, for example, from 60 to over 10,000 cycles, with the light output increasing almost linearly with frequency in this range. Figure 3 shows a log-log plot of the light output as a function of voltage for a fixed frequency of 420 cycles/sec. The slope shows the light output to vary roughly as the third power of the applied voltage.

The photoconductor is a CdS powder also bonded with plastic. Such powders were developed a few years ago by Dr. S. M. Thomsen[9] of the RCA Laboratories. Compared to other photoconductors, this powder has the advantage that it can easily be formed into uniform layers of arbitrary

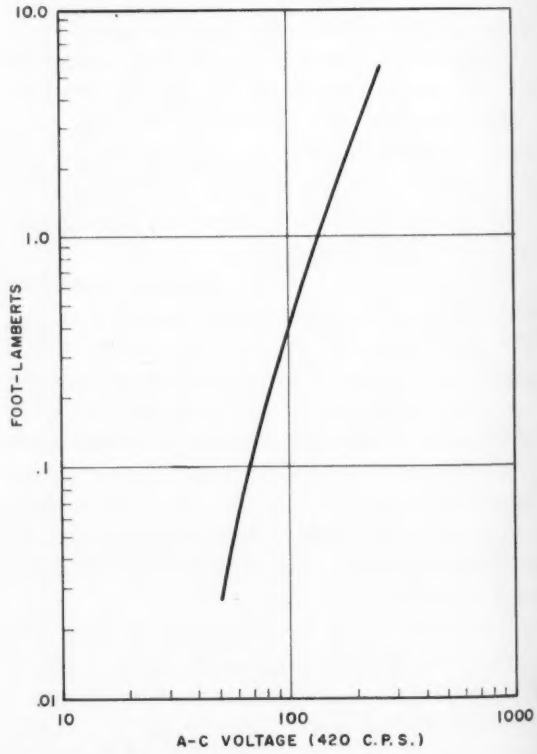

Fig. 3. Phosphor output light versus applied voltage

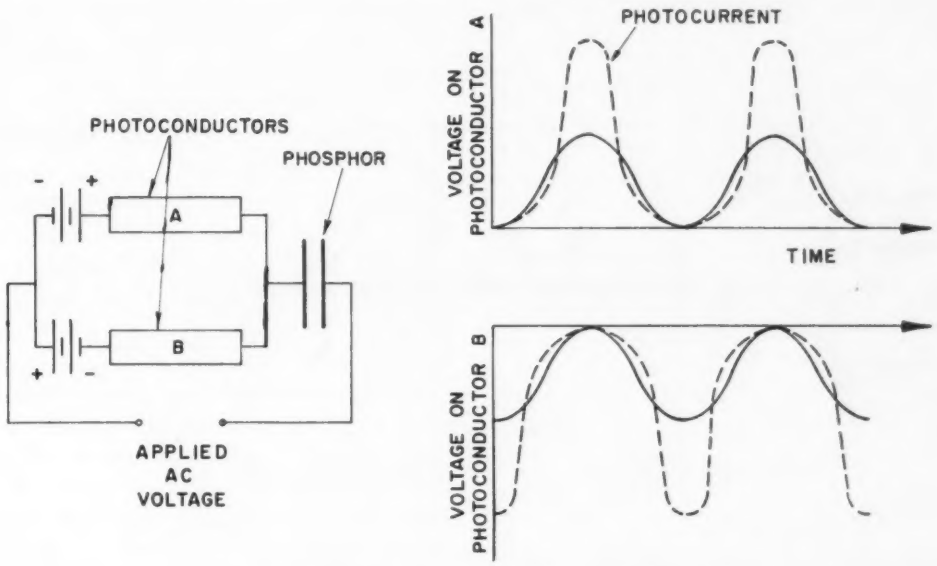

FIG. 4. Amplifier element with biased a-c operation.

size and thickness.[7] In addition, it has a sensitivity to roentgen rays and light comparable to the best photoconductive materials.

Although the photoconductive powder can be operated with a-c voltage, its sensitivity is as much as 10 times greater when operated with d-c or pulsed d-c voltage.[4,5] Since the phosphor layer requires a-c voltage, being inoperative with d-c, this difference cannot be reconciled by the simple series circuit shown for the double-layer type of intensifier. One method of overcoming the problem is shown in Figure 4 representing a single amplifier element. Instead of a single photoconductor, two smaller photoconductors are used, both of which are assumed to be equally irradiated. In series with the photoconductors are bias voltages of opposite polarities, and across the entire combination is an a-c voltage. Since the bias voltages are equal to the peak a-c voltage, pulsating d-c voltages of opposite polarity are produced as indicated to the right. Because of the very nonlinear current-voltage characteristic of the photoconductor, the current pulses are narrow, as indicated by the dashed curves. Since the current pulses are of opposite polarity and phase, the phosphor element is charged positive during one half cycle and negative during the next half cycle. In this way the phosphor is excited with a-c while pulsating d-c voltage is used to obtain high sensitivity from the photoconductor. In practice two rectifier elements can be substituted for the d-c supplies to obtain the pulsed d-c. Such operation is almost as efficient, requiring only an a-c voltage source.

To use the pulsed d-c method of operation, the amplifier design shown in the cross section of Figure 5 is employed. For illustrative purposes the thickness of the layers is greatly exaggerated. As a base a glass plate is used, the upper surface of which has a transparent conducting electrode. On this surface a thin layer of electroluminescent phosphor is provided. On the side facing the roentgen-ray beam is the photoconductive layer which has grooves cut into it and narrow conducting lines along the top. Between the phosphor and photoconductive layers are two other auxiliary layers. One is a thin opaque insulating layer to block feedback light from the phosphor. The other, called the current-diffusing layer, has a conductivity sufficient to allow currents to flow from the photoconductor to the phosphor layer and

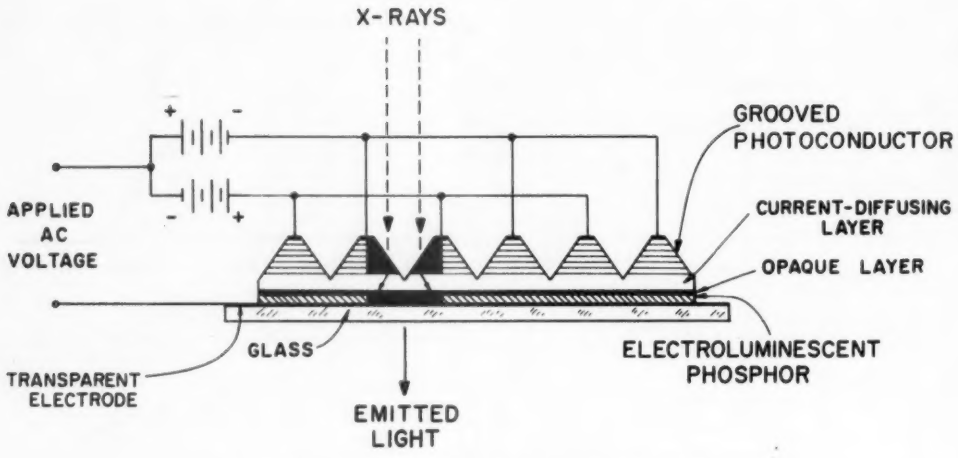

Fig. 5. Cross section of amplifying fluoroscope screen.

from the base of a photoconductive ridge to its neighbor. At the same time the conductivity is not great enough to allow spreading of the photocurrents more than about one groove width.

By connecting the conducting lines into two interdigital groups, opposite polarity voltage pulses are applied to adjacent photoconductor ridges. The darkened areas show a single picture element excited, with the two irradiated portions of photoconductor and the associated phosphor area corresponding to the single element of the preceding figure.

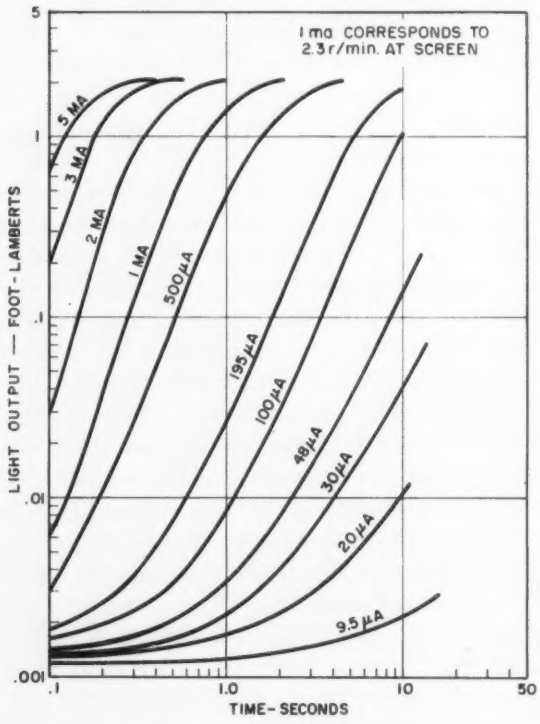

Fig. 6. Light output versus time for various roentgen-ray currents.

Input-Output Characteristics

When excited with a roentgen-ray image, the amplifier does not respond instantly but has a build-up characteristic due to the photoconductive material. Figure 6 shows build-up curves of the amplifier output as a function of time for different levels of roentgen rays. These show that the time to reach a specified light level decreases as the roentgen-ray level is increased. For a given time of excitation, the output light can be determined as a function of input roentgen-ray level from these curves. Figure 7 shows an input-output characteristic for a ten second excitation of the amplifier. The input level is shown in terms of roentgen-ray current and also milliroentgens per minute. For comparison, the output light of a Patterson Type CB-2 screen is also plotted. The 45 degree slope of the Patterson screen indicates its linear output. The much higher slope of the amplifier characteristic indicates it to have a gamma of about 3.

These curves show the amplifier output to be as much as 100 times brighter than the Patterson screen for equal levels of roentgen-ray excitation.

Examination of the amplifier characteristics with other exposure times shows that the output light level reached is approximately determined by the ma.-sec. product of the roentgen-ray source. Figure 8 shows the amplifier characteristic in terms of the ma.-sec. input with a target-to-amplifier distance of 24 inches and a 0.5 mm. Al filter. The equivalent exposure of the amplifier in milliroentgens is also shown along the abscissa. The horizontal lines through the curve show the range of experimental variations in input for a given output (neglecting the 9.5 μa. and 20 μa. curves of Figure 6). As shown, an exposure of between 5 and 50 mr is required to excite the amplifier. By comparison, about 60 mr is required to obtain a density of 1 on Kodak Blue Brand medical film.[11] At the conventional levels used in posteroanterior fluoroscopy of a chest about ten seconds of exposure are required for image build-up on the amplifier. The high gamma of the amplifier and its latitude are similar to Eastman Blue Brand medical film used with calcium tungstate screens. The curve of this film is shown in Figure 9 for comparison, with the density and exposure in relative values. The straight-line portion similarly has a high gamma, and the latitude is also about 10.

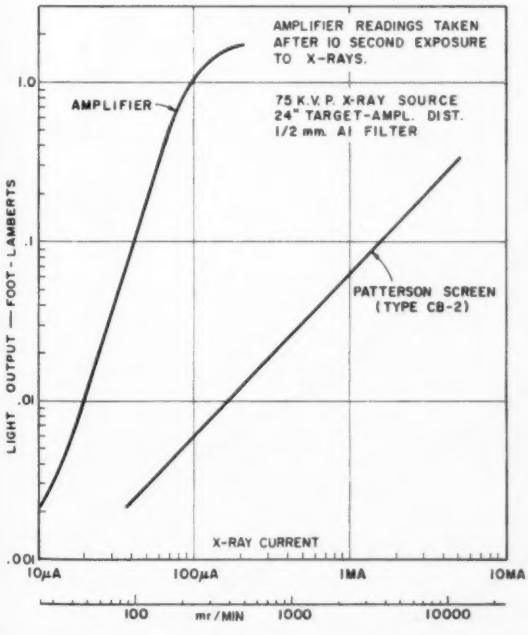

Fig. 7. Input-output characteristics of amplifier and Patterson screen.

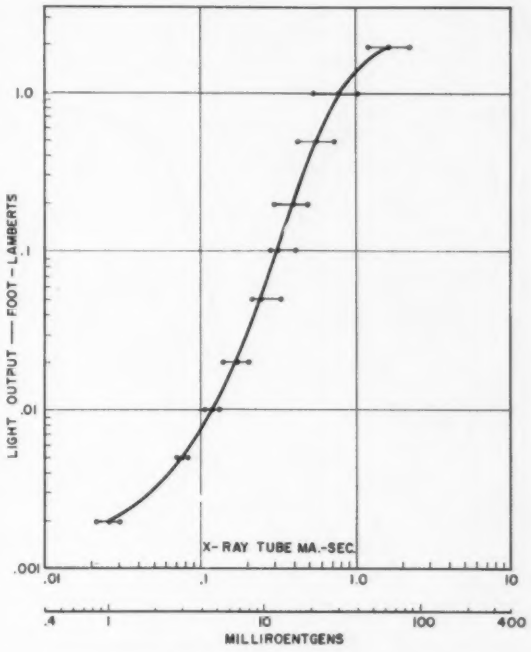

Fig. 8. Light output versus roentgen-ray dosage on amplifier.

DECAY PROPERTIES

In addition to its build-up, the amplifier has long decay properties. This is shown by the curves of Figure 10 obtained by exciting the amplifier for a one second period with different roentgen-ray levels. These curves show the amplifier output to decay slowly over a fifteen to thirty second period. Because of internal heating of the photoconductor, the 200 mr curve, shown dashed, has a more rapid decay rate. The time of decay depends on the photoconductive material, in some instances being in the order of minutes. Because of this long decay, a picture previously excited can be viewed with the roentgen rays cut off. In such cases a fixed image can be observed

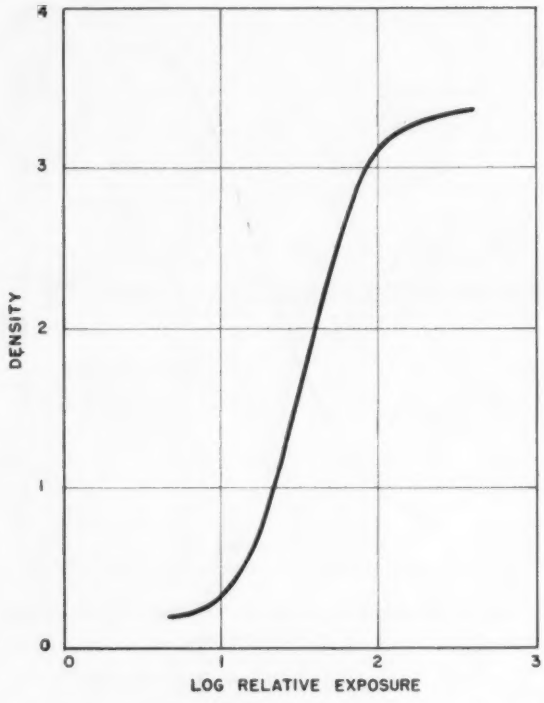

Fig. 9. Characteristic curve of Eastman Blue Brand roentgenographic film with calcium tungstate intensifying screens.

for an extended time with a saving in dosage of roentgen rays. If the object being roentgenographed is not stationary, a short pulse of roentgen rays can be used to produce a "temporary photograph" for extended viewing. Since, in addition, the high-lights of the output picture may exceed 1 ft.-lambert, the image can be comfortably viewed with moderate room illumination.

Because of the long decay of the bright image, the time-integrated light is relatively high. This is of interest in applications where the output of the screen is to be photographed. From the decay curves the time-integrated light was determined, and in Figure 11 this integrated light is shown as a function of roentgen-ray input in milliroentgens. By comparison, the integrated light output produced from a Patterson CB-2 screen with the same excitation is also plotted. This shows the integrated output of the amplifier to be about 100 times that of the Patterson screen at inputs of about 10–20 mr. However, because of the high

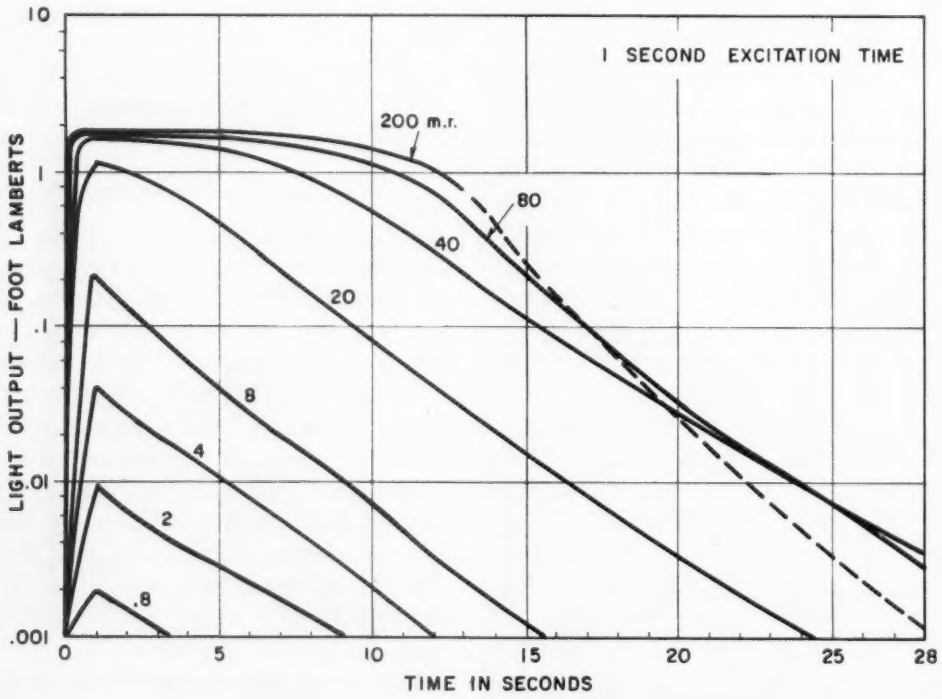

Fig. 10. Build-up and decay for various roentgen-ray input levels.

gamma of the characteristic and the rapidly decreasing output of the amplifier at low roentgen-ray exposures, its advantage at these levels is reduced. With more sensitive materials and longer decay, it is hoped that the amplifier can be used for reducing the roentgen-ray dosage in photofluorography.

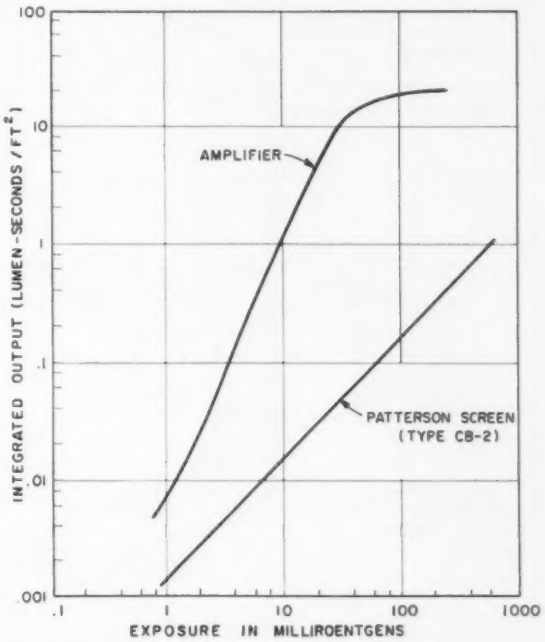

FIG. 11. Time-integrated light output of amplifier.

IMAGE ERASING

Unless special erasing means are used, the long decay of the photoconductor prevents the viewing of a new image for a period of many minutes or more. If the photoconductor is not allowed to decay sufficiently, the new image will re-excite portions of the old image. However, by using a special property of the photoconductive powder, erasure of an old image can be accomplished in a fraction of a second at any desired moment during the decay. The panel can then be used instantly for viewing a new image.

In Figure 12 is shown the d-c decay curve of a photoconductive sample initially excited for a one-second period. Although the decay current is shown up to one hundred seconds, the current continues to drop slowly beyond this time. To reach the level of the lower dashed line an hour or more is required. If instead of allowing the photoconductor to decay normally, the applied

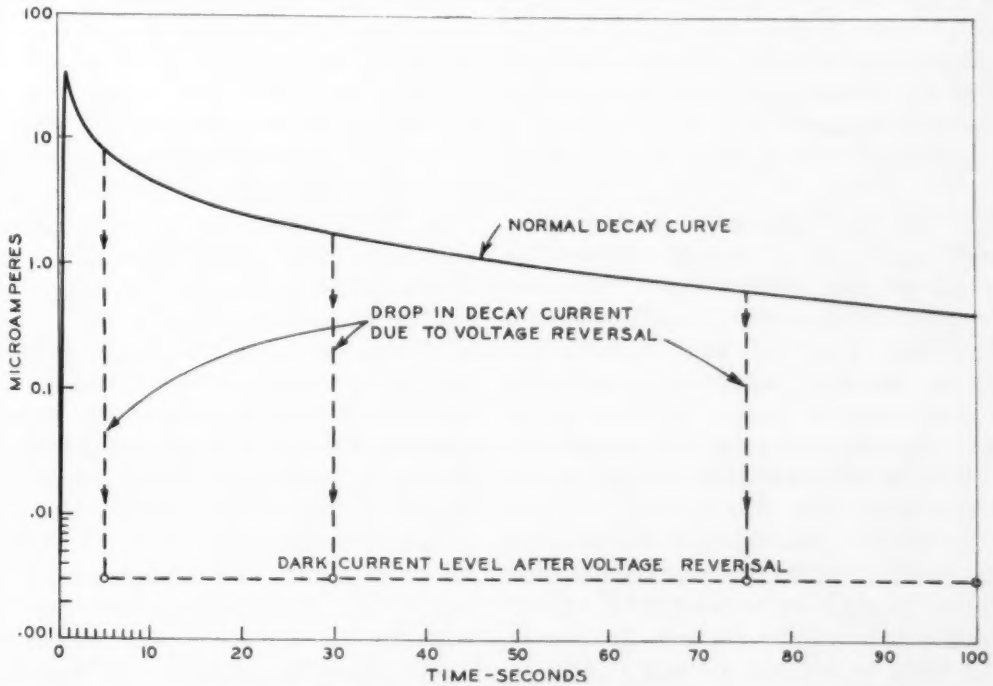

FIG. 12. Effect of voltage reversal on photocurrent decay.

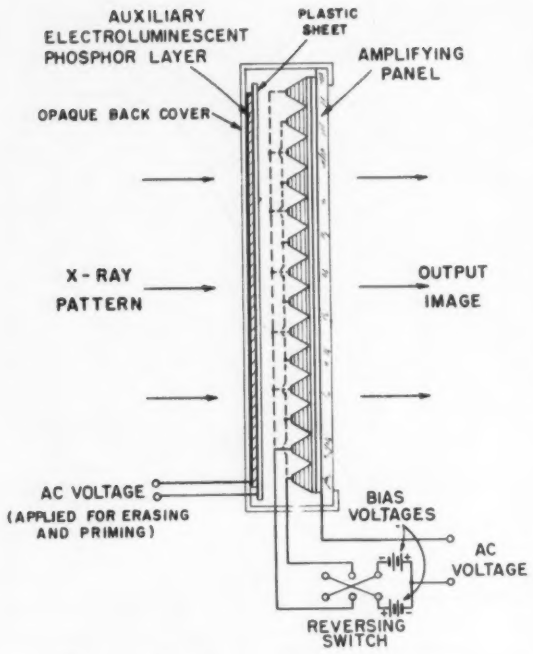

FIG. 13. Arrangement for erasing image.

voltage is suddenly reversed, the photocurrent (neglecting the polarity change) instantly drops to a low level many orders of magnitude less than its normal level. As shown, the voltage can be reversed at an arbitrary time during the decay, driving the photocurrent down to the same low current level. Immediately after being erased the photoconductor can be excited again with a roentgen-ray signal to a new current level.

This type of erasing can be applied to the amplifying panel in several ways. The simplest method is to reverse the polarity of the bias voltages on the panel at the moment erasure is desired. With reversed polarity the panel can be then excited with a new roentgen-ray image. For successive images, this procedure can be continued.

In the voltage-reversal process a relation exists between the photocurrent reached during excitation and the dark current level which results after voltage reversal. In some cases the higher the photocurrent the lower the dark current after erasing. Because of this, erasing by voltage reversal alone may be nonuniform or incomplete. An improved operation is possible with the arrangement shown in Figure. 13. Here an auxiliary, thin electroluminescent layer on a plastic sheet is provided on the inner surface of the light-tight back cover of the amplifier. In operation, the roentgen-ray image passes through the cover and the auxiliary phosphor layer. Since the phosphor layer is about 1 mil thick, its attenuation of the roentgen rays can be neglected. After the amplifier has been excited by the roentgen-ray image and the picture viewed, erasing is accomplished by reversing the bias voltages connected to the photoconductive grooves and simultaneously switching on the auxiliary phosphor to flood the photoconductor for a fraction of a second. The bias voltages are then reversed again, leaving the photoconductive layer at a uniformly low dark level over the entire surface. The amplifier is now ready to be excited with a new roentgen-ray image. In some cases the dark current level is so low after erasure that the panel has reduced sensitivity for a new image. This can be overcome by a priming action whereby the auxiliary phosphor layer is momentarily switched on again during or just preceding the excitation with the new roentgen-ray image.

EXCITATION BY FLUOROSCOPE SCREEN

Since the photoconductive layer is sensitive to visible light as well as roentgen rays, the amplifier can be used to intensify the visible image of a fluoroscope screen. To avoid optical losses, the fluoroscope screen can be placed in contact with the photoconductive layer or the phosphor material can be deposited in the groove spaces of the photoconductor. Tests with the Patterson CB-2 screen and the amplifier indicate that about the same gain or slightly less is obtained by this method of operation compared to exciting the photoconductor. As shown in Figure 14 the peak sensitivity of the grooved photoconductor is at about 750 mμ. in the deep red, while the peak emission of the phosphor is about 550 mμ. in the green. The value of this type of operation

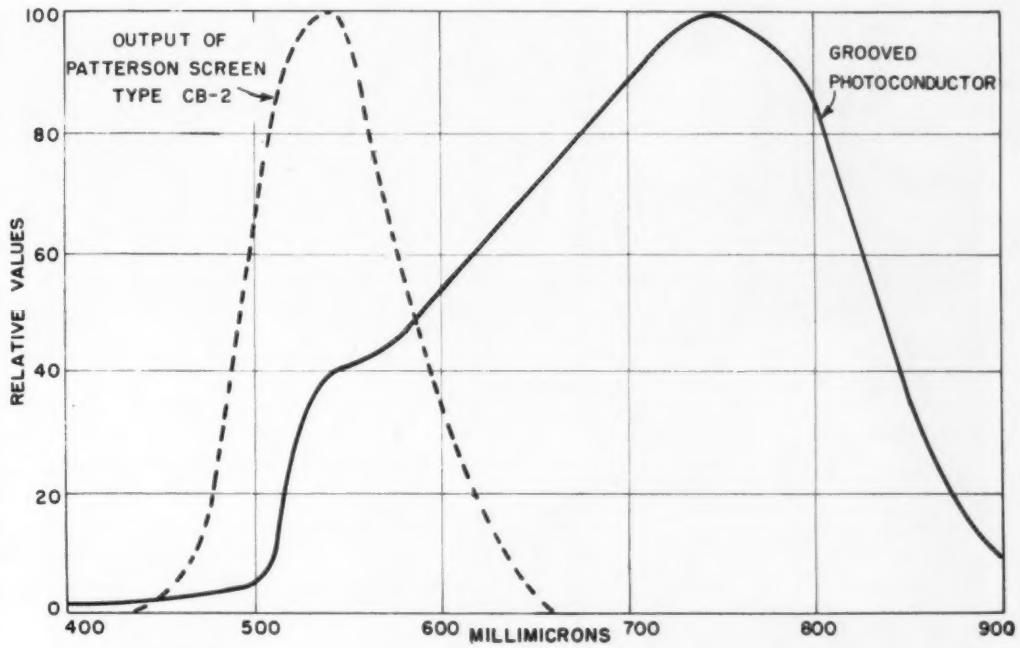

Fig. 14. Spectral response curve of grooved photoconductor.

is dependent on the future availability of better matched new materials having comparable efficiencies.

Resolution and Contrast Sensitivity

The resolution of the amplifier is limited by several factors. Since the photoconductive layer has grooves cut into it with a 25 mil spacing between centers, picture details smaller than this cannot be observed in the direction across the grooves. In order to absorb a large fraction of the roentgen rays and to efficiently control the output phosphor, the photoconductive layer is made about 12 mils thick. This results in spreading of the photocurrents by roughly this amount along the grooves. Some loss in resolution is also caused if the current-diffusing layer has too high a conductivity. It is believed, however, that with improved design, amplifier panels can be made comparable in resolution to conventional fluoroscope screens.

Because of its high gamma, the amplifier is potentially useful for detecting small variations in object thickness or density. However, with the experimental fabrication techniques presently used, nonuniformities in amplification over the surface limit the contrast sensitivity. With $\frac{1}{2}$ inch thick aluminum, large-area variations in thickness of about 6 per cent can presently be detected. It is believed that with improved techniques, lower limits can be reached.

The output of a roentgen-ray-excited 12 inch amplifier panel is shown in the photograph of Figure 15. Immediately above the amplifier is a Patterson CB-2 screen simultaneously excited with the same roentgen-ray level. Because of its relatively low output, it is entirely dark in the photograph. For producing the image a 5 inch thick phantom of pressdwood sheets was used between which were inserted a femur containing a Smith-Petersen nail, a hypodermic syringe, and a simulated section of intestine made of $BaSO_4$ in plastic. In addition, a number of metallic objects were inserted, some of which extend into the area of the Patterson screen. The distal half of the femur was cut off and placed above, allowing the shaft to extend also across the Patterson screen. A thin amplifying screen

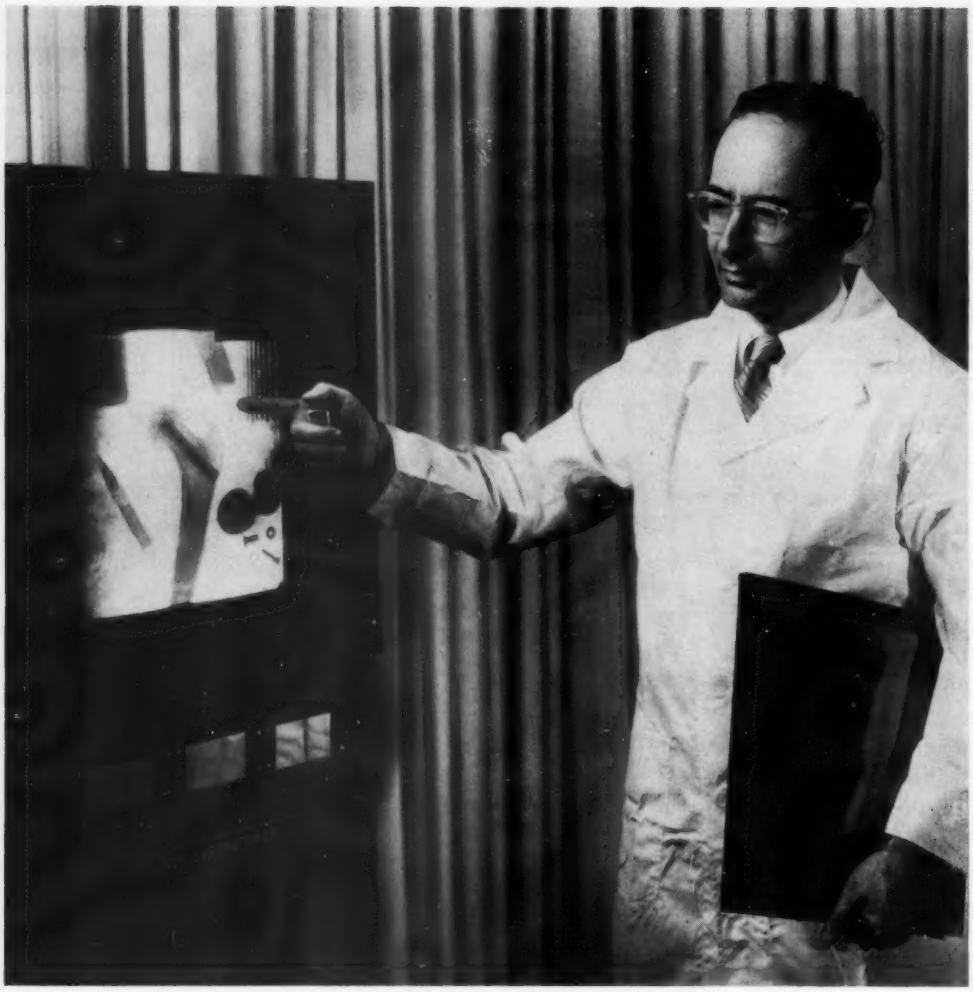

Fig. 15. Photograph of amplifier screen in operation.

identical to the one shown in operation is held by the author.

SUMMARY

The amplifying screen requires approximately the same roentgen-ray exposure as Kodak Blue Brand medical roentgenographic film. Compared to the conventional fluoroscope screen, the output image is about 100 times brighter and has a gamma 3 times higher. In posteroanterior fluoroscopy of a chest, for example, an image of about 0.01 ft. lamberts on a conventional screen is increased to about 1 ft. lambert on the amplifier. At the same time 2:1 variations in brightness of image details are increased to almost 10:1 variations by the amplifier. With its long persistence or storage, the amplifier permits a stationary output image to be viewed for an extended time with the roentgen rays off. This enables a reduction in dosage of several times or more.

Solid-state amplifying panels are relatively new devices. Although an accurate estimate of their ultimate possibilities is difficult, it is hoped that continuing research in the future will bring sufficient improvements so that they can be used for the viewing of moving objects. For this purpose, however, several orders of magnitude of improvement in materials are possibly

required. In addition to the long-term objectives, it is also hoped that the relatively smaller improvements occurring along the way will enable such amplifiers to gradually find uses in specific applications.

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EDITORIAL

THE LIFE HISTORY OF CRANIAL VAULT SUTURES AS REVEALED IN THE ROENTGENOGRAM

IN THE history of our knowledge of cranial sutures, fact and fancy are closely interwoven. In the middle of the sixteenth century, Vesalius and his pupil Fallopius pointed out correctly that sutures undergo progressive obliteration with advancing age.[2] Among the more fanciful and speculative suppositions, we find statements that early suture closure indicates a more primitive type of brain, that the more the brain is exercised the longer the suture remains open, and that in the "lower" races the sutures are simple and commence to fuse in the direction from anteriorly to posteriorly, while in the "higher" races the sutures are more complex and the order of fusion is reversed. Gratiolet therefore spoke of "frontal" and "occipital" races.[20]

None of these statements has withstood the test of probing investigation. As far as racial differences in the date of suture closure are concerned, Todd and Lyon[31,32] disproved the assertion that cranial sutures in white people fuse later than in Negroes. In their extensive material they found no significant differences.

For the radiologist the state of the sutures of the cranial vault is of practical importance mainly for three reasons: the roentgenographic appearance of the sutures may be confused with pathologic findings, particularly a fracture dehiscence; the evaluation of the sutures may be of assistance in the determination of the chronologic and physiologic age of the individual; and finally their obliteration may be accelerated, as in premature synostosis, or abnormally delayed as in cretinism and hydrocephalus.

In a recent extensive paper Crocellà[6] has dealt with the roentgenographic demonstration of the sutures. By introducing a radiopaque suspension of barium sulfate into the sutural gap of macerated skulls and by roentgenographing the latter in the three axes of the skull, he systematically studied the roentgenographic appearance of the important sutures and defined optimal projections for each of them. He includes the submentovertical view among the desirable projections.

Stereoroentgenography or slight angling of the head in profile view will display the coronal and lambdoid sutures of both sides. So far laminagraphy seems not to have been utilized for better definition of the sutures. The occipital or Towne projection shows the coronal and lambdoid sutures to advantage[21] and is essential for demonstration of intersutural or wormian bones.[24]

In the roentgenogram of young infants, the bones of the cranial vault are separated by translucent clefts which in the normal, at the end of the first year of life, have reached a width of not more than 0.5 to 1 mm.[14,19] The sutural edges of adjacent bones are at first smooth, and the suture appears as a narrow translucent line which widens toward the fontanelles. Later it becomes wavy and small projections appear from the margins of the bones. As these jagged edges start to interlock, they send out a second series of projections, and finally a third, giving the sutures a markedly serrated appearance. The less jagged condition of the sutures therefore corresponds to an earlier stage of development.[26] The serrations appear well formed during the third year of life. It is most important to realize that the dentations are found only

in the outer table of the calvarial bones, while at the inner table the suture is represented by a thin translucent line. The latter may appear roentgenographically in unfused sutures as a translucent marking that can be traced as a straight or slightly wavy line through the complicated meandering of the external suture. Leaving aside this exception which appears only on favorable roentgenograms, it is the ectocranial part of the suture that is roentgenographically demonstrable.[14]

As age progresses to maturity, the appearance of the sutures shows slight changes. The suture line narrows and its dentated margins become more compact and sclerosed. This parasutural hyperostosis is particularly well marked in the upper portion of the coronal sutures,[18] but sometimes can be found also elsewhere. Caffey[5] warns against mistaking this clinically insignificant feature for a pathologic hyperostosis accompanying a neoplasm. Occasionally, an unobliterated sagittal suture, when struck tangentially on profile view, will give a peculiarly moth-eaten, striated appearance, which easily can be mistaken for a destructive bone process.[14]

The beginner and occasionally also the experienced must guard against mistaking the roentgenographic appearance of the sutures for a traumatic separation. The following locations offer the most common pitfalls: the separate, fissure-like translucency of the inner suture line projected into or besides the outer line,[3,7] the slight depression at the height of the coronal suture,[18] the occasional overlap of the occipital bone in relation to the parietal bone at the highest point of the lambdoid suture,[24] the absence of dentations in the lower portion of the coronal suture and the resulting straight or only slightly wavy appearance of the suture line in this area,[22] rarer and less well known suture lines that regularly appear in the infant, but only uncommonly persist into adult life such as the mendosal suture in the occiput and the metopic suture in the frontal bone,[5,19,21] and finally the coronal and sagittal suture lines as presented on axial view roentgenograms.[11]

After the skull has reached its definitive size, a date that coincides with the attainment of maturity in the rest of the skeleton, the sutures start to obliterate. Fusion seems to begin in the diploe, to be followed by obliteration of the suture in the lamina interna and finally in the externa.[14] On the basis of their gross studies, Todd and Lyon[29,30] contend that suture closure starts upon the endo- and ectocranial surfaces of the skull simultaneously, but that ectocranial closure progresses more slowly and shows more individual variation.

Three broadly conceived gross anatomic studies on suture fusion have been undertaken by Frédéric[10] and by Davida[8] on the continent and by Todd and Lyon[29,30 31,32] in this country. These authors subdivide each suture into smaller sections, and assess the state of fusion individually. They also assign numerical values to indicate the degree of fusion, from o for the open suture to 4 for complete obliteration. They are in agreement that fusion at the outer and inner tables should be evaluated separately. For the male series they establish the following sequence of fusion of the vault sutures: sagittal, coronal and lambdoid. They also note that the part of the sagittal suture between the parietal foramina, the so-called pars obelica, is the first to obliterate. The sequential pattern in the female is less regular, with the coronal suture being the first to ossify in one group of cases.[8]

As for the actual dates of suture closure Todd and Lyon[29,30] have given the most definite information which has become the standard reference. The greatest activity in suture closure occurs between the years of twenty-six to thirty. There are, however secondary periods of obliterative activity in the later thirties and at about sixty years, trying to complete what has been missed during the earlier spurts of fusion activity. Failure of complete union is considerably more frequent in the sutures of the cranium than in the epiphyses of the long bones.

In recent years Todd and Lyon's work

has been subjected to some distinct criticism.[1,4,28] Singer[28] points out, that Todd and Lyon used only male skulls and eliminated all specimens which showed "irregularity of closure." Their data therefore represent a select group from which "by arbitrary rejection of awkward material"[1] unsuitable cases were excluded.

What is the value of the study of suture closure for estimating the age in a given individual? Here the pendulum of scientific opinion has vacillated from complete acceptance to rejection. Frédéric[10] concluded that it is not possible to gauge the age of any particular individual more closely than within one decade. Todd and Lyon[29,30] intended to improve this by narrowing the range to six years but, as they themselves concede, were not too successful in individual cases. The anthropologist Hrdlicka[15] regards age determination from the state of the cranial sutures useful when taken conjointly with other age characteristics. Recent studies by Franchini,[9] Brooks,[4] and Singer[28] have confirmed Hrdlicka's axiom. All authors agree that age estimates based on cranial suture closure can be utilized only if taken in conjunction with other indicators of adult age since the phenomenon itself is too erratic.

In the female group the discrepancies are still greater. Here we find the tendency for vault sutures either to remain open throughout life or to close much later than available prediction tables indicate.[4] However, although the method may fail in an individual case, this does not invalidate the contention that for larger groups the average age might be computed quite accurately from suture studies.

The roentgenologic method is, of course, particularly adept in utilizing large numbers of cases. Paralleling the roentgenologic investigation of developmental stages in the hand and knee,[13,23] cross-sectional or horizontal studies for different age groups need to be undertaken as well as longitudinal explorations, which follow individuals through their lifespan by means of roentgenograms of the skull. Furthermore, roentgenographic suture studies need to be correlated with clinical and laboratory data including roentgenograms of the extremities in the younger age groups. So far such correlations have never been attempted, since most investigations have been made on skeletal material where the investigator regarded himself as fortunate if information on sex and racial origin was available. Roentgenographic serial studies in cases of acceleration or delay of suture closure may furnish a key to the understanding of these conditions, if such studies include evaluation in terms of normal roentgenographic standards for the respective age groups and also take account of pertinent clinical and laboratory data. Serial roentgenographic examinations may also give the best indication for clinically warranted surgical interference in premature synostosis.[27,34]

Roentgenographic investigations on twins, siblings and consecutive generations would open up the field of sutural anatomy to the genetic approach. A beginning has been made by studies of Greene[12] on rabbits, of Ingraham and his co-workers[16] on the presence of cranial synostosis in siblings of two generations, and particularly by the roentgenographic studies of Torgersen.[33] The latter showed that the persistence of the frontal or metopic suture, which otherwise disappears between the ninth month of infancy and the third year,[25] is a hereditary dominant trait with varying penetrance, that could be demonstrated in 8 pairs of twins and in one family in 3 generations. The presence of the metopic suture is an indicator of a predisposition to late closure of other sutures and to the persistence of abnormal sutures, as exemplified by the frequent occurrence of sutural bones and by an increased variability of the sutural pattern. Torgersen[33] comes to the conclusion that there are probably two groups of genes influencing the sutural pattern of the skull, one determining the site of the cranial ossification centers and location of the sutures and another affecting the rate of suture obliteration.

In this discussion on roentgenographic

verification of suture closure, it is all-important to realize that the roentgenologic results cannot be compared directly with anatomic standards as developed by study of the bones of the skull themselves. Recently, Keen[17] again called attention to the fact that anatomic and roentgenographic criteria for age determination cannot be reconciled and must be treated as separate entities. More than twenty-five years ago Haas[14] in a classical paper on clinical applications of the roentgenologic suture diagnosis pointed out that a roentgenologically unfused suture is not necessarily anatomically patent, since the roentgenographic diagnosis is based on the presence of an open suture line in the lamina externa only. Obliteration of the suture only in the lamina interna would be regarded anatomically as a partial synostosis; it would be evaluated for clinical purposes as complete closure, but would be identified as an open suture on the basis of the roentgen evidence, since the serrations in the lamina externa would still be demonstrable.[14]

In a practically unknown, but important and very carefully executed thesis, Manns[20] has attempted to appraise suture closure exclusively from the roentgenologic point of view. The author utilized posteroanterior, anteroposterior and profile views in more than 1,500 cases of known age and sex including all age groups. She classified the sutural status as open, partially closed and closed, and arrived at the following conclusions. Roentgenologically, fusion starts in the coronal suture, followed by the sagittal and lambdoid sutures in this order. The coronal suture is completely obliterated in only 6.2 per cent of the males and 1.8 per cent of the females in the age group of twenty-one to thirty years. It shows partial or complete obliteration in the age group of thirty-one to forty years in 80.2 per cent of the males and 45.3 per cent of the females. It is always fused at old age. The sagittal suture is totally obliterated in 4.9 per cent of the males and in none of the females in the age group of twenty-one to thirty years. It is always closed in men aged sixty-one to seventy years and in women aged fifty to sixty years. The lambdoid suture is only exceptionally closed in men aged thirty-one to forty years. It is obliterated in 14 per cent of the males and 10 per cent of the females, aged forty-one to fifty years. One third of all males and 30 per cent of all females aged seventy-one to eighty years still display an open lambdoid suture.

In summary, it can be concluded that in the roentgenologic investigation of cranial vault sutures we have a means of evaluating a facet of physiologic differentiation throughout life and at ages where most other skeletal indicators have lost their usefulness. In common with other life processes, obliteration of sutures seems to be affected by genetic, metabolic and hormonal factors.

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THE LAWRENCE REYNOLDS LIBRARY

ON SUNDAY afternoon, February the Second 1958, The Lawrence Reynolds Library was dedicated by impressive ceremonies at the University of Alabama Medical Center in Birmingham. This Library, which contains one of the world's finest and most valuable private medical collections, was donated to his Alma Mater and his native state by Dr. Lawrence Reynolds, Editor of this Journal, who spent a lifetime acquiring it.

This rare collection is housed in a special wing which the University has built as an extension to its new Medical Center Library. It consists of nearly 6,000 individual pieces of original manuscripts, autographed letters and old medical books, some of them dating back to the fifteenth and sixteenth centuries, representing samples of the highest achievement in Renaissance printing.

The section of original manuscripts includes, among others, the fourteenth century manuscripts of Arnoldus de Villa Nova entitled *Regimen Sanitatis ad regem Aragonum* and John of Toledo's *Dietarium;* the original manuscript of the article on *The Stereoscope and Stereograph* contributed to the *Atlantic Monthly* in June, 1859 by Oliver Wendell Holmes; and of the more recent ones the address on *The Discovery of Radium* by Mme Marie Curie (Sklodowska) delivered on May 14, 1921 at Vassar College, Poughkeepsie, New York, with an autographed photograph.

The letter section contains all the correspondence of Louis Pasteur with the Mayor of the village on silk worm disease, consisting of 20 holographic letters of Pasteur (1866–1889), 8 letters in the hand of Mme Pasteur, and 2 unpublished photographs; the letter of Louis Pasteur to Monsieur Marchand, dated August 12, 1881, on rabies; and other items from the pen of this giant of science who revolutionized both industry and medicine by applying laboratory knowledge to fight disease. There are also signed letters with typed transcripts by Sir William Osler to his "good friend Ned Milburn" (1865–1919), and two original letters from Wilhelm Conrad Röntgen and Frau Wilhelm Conrad Röntgen to Louis Grauel; and others.

Of the large section of medical books of the sixteenth and seventeenth centuries, three groups deserve special mention. It is now generally agreed that the foundations of modern medicine were laid down in this period by three epoch-making contributions. In 1543 the first complete text book on human anatomy was published by Andreas Vesalius entitled *De humani corporis fabrica librorum epitome*, Johannes Oporinus, Basel. This book, as well as Vesalius' other contributions, was illustrated with magnificent drawings by van Calcar, a pupil of Titian. The Library contains not only Vesalius' textbook in its first edition but also other works of the truly great anatomist, several later editions of his works and a group of books on the works of his teacher, Sylvius, who from a friend turned into a bitter foe, of Fallopius and Eustachius, his successors, and of other famed anatomists of the period, most of whom became "Vesalius converts." This study of the "fabric" of the human body based on pure research was soon followed by an interest based on similar ground in the functions of the human body. Among those who pursued such a course was Andreas Caesalpinus, who, in his *Peripateticarum Questionum Libri Quinque* published in Venice in 1571, began speaking of the "circulation" of the blood. His bold theory, however, was not supported by experimental proof and thus he had no influence on his contemporaries. Later in 1628, at the book fair in Frankfurt, a slender, poorly printed volume was displayed by William Harvey on the *Exercitatio anatomica de motu cordis et sanguinis animalibus*, presenting for the first time experimental proof that the heart acts as a pump causing circulation of the blood throughout the body. The books of Caesalpinus and Har-

vey and 12 other texts and facsimiles of the book of Harvey published in different countries form the nucleus of the second group of works, containing the contributions of Fabricius Hieronymus of Aquapendente, Harvey's teacher, on *De venarum ostiolis;* of Francis Bacon, one of Harvey's most notable patients, on *Instauratio magna* (*Novum organum*); Harvey's second great work *Exercitationes de Generatione Animalium* (1651) on embryology and many similarly rare items. The third group of collections of this period is highlighted by works of Ambroise Paré, the greatest surgeon of the sixteenth century. Paré based his practical surgery on the new concepts expounded in Vesalius' *De Fabrica* and, after being able to conquer the prejudices of the time, introduced sound methods for the treatment of a rather significant number of lesions. His influence on contemporaries and successors for the next two centuries is amply reflected by the many contributions comprising this group.

In 1600, William Gilbert published his epochal work *De magnete, magneticisque corporibus et de magno magnete tellure; physiologia noua, plurimis argumentis experimentis demonstrata*, establishing the basis for the formidable sciences of magnetism and electricity, which were to develop. The early progress was rather slow until the middle of the eighteenth century when Benjamin Franklin in his extensive *Experiments and Observations on Electricity Made at Philadelphia* defined "positive" and "negative" electricity and considered, among many other things, the possibility of curing palsy by electric shock. In 1786 Luigi Galvani discovered "animal electricity." This was followed in rapid succession by Alessandro Volta's construction of the first electric pile, as communicated in a letter to the Rt. Hon. Sir Joseph Banks (1800); by Hans Christian Oersted's *Experimenta circa effectum conflictus electrici in acum magneticum* (1820), establishing a link between electricity and magnetism; by André Marie Ampère's *Mémoire présenté à l'Académie Royale des Sciences sur l'action mutuelle entre deux courants électriques* (1820), describing the mutual effect of two electric currents; and by G. S. Ohm's work in 1827, formulating the relationship between voltage, amperage and resistance. Then in 1837, Faraday and Henry discovered electromagnetic induction. These important works and many others in the chain of development of the two sciences form an eminent part of the Library and constitute a monumental introduction to the vast collection of more recent contributions in the field of radiology.

Among the more recent contributions are also many rare works from other branches of medicine, as exemplified by some 40 writings from the pen of Sir William Osler, the great physician of modern times; an almost complete set of Harveian Orations, beginning in 1865; an extensive compilation of Nobel Prize Papers; and others.

These are but brief heuristic glimpses into a magnificent Library. It is written on the dedication plaque that graces its entry: "Each time one of you reaps from the great minds of the past the desire for finer achievement in your profession and nobler development of your own character, The Reynolds Library will have been rededicated." In the ancient words of Terentianus Maurus spoken in 1286, *Pro captu lectoris habent sua fata libelli.*

Traian Leucutia

BOOK REVIEWS

Books sent for review are acknowledged under: Books Received. This must be regarded as a sufficient return for the courtesy of the sender. Selections will be made for review in the interest of our readers as space permits.

THE HEAD AND NECK IN ROENTGEN DIAGNOSIS. Volumes I and II. By Eugene P. Pendergrass, M.D., Professor of Radiology and Chairman of the Department of Radiology, University of Pennsylvania; J. Parsons Schaeffer, M.D., Ph.D., Professor of Anatomy and Director of the Daniel Baugh Institute of Anatomy, Emeritus, Jefferson Medical College; and Philip J. Hodes, M.D., Professor of Radiology, University of Pennsylvania. Second edition. Cloth. Price, $37.50. Pp. 1,759 (plus indices), with 1,740 illustrations. Charles C Thomas, 301 East Lawrence Avenue, Springfield, Illinois, 1956.

This monumental work in two volumes, representing a revision of the now famous original "The Head and Neck in Roentgen Diagnosis" by Pancoast, Pendergrass and Schaeffer, is indeed a superlative production. There is an abundance of illustrations in these two volumes, many of them re-made and others entirely new, to amplify the text. For the most part they are of excellent quality and very informative. Many of them are based on actual anatomic studies and the roentgen anatomic application. All of the new techniques are presented with their anatomic background, making it possible to clearly correlate anatomic features with the shadows seen on the roentgenograms. It may be said that the keynote of the book is correlation of demonstrated anatomic features with their roentgenographic appearance.

The current edition has been divided into sixteen chapters with eight in each volume. The gamut of possible subjects has been exhaustively covered, including detailed roentgen anatomy of the skull in both its normal and pathologic variations, as well as the normal and pathologic roentgen anatomy of the cervical spine. A most complete index at the end of each volume makes the myriad details readily accessible for quick reference.

The entire text has been written in an easy conversational style, preventing what could become a tedious exercise in the accumulation of detailed factual knowledge. The authors have covered by adequate discussions all of the rather rare conditions, as well as the common conditions, which could possibly be enumerated in each section. This feature alone makes the book an excellent work of encyclopedic scope. Conveniently arranged at the end of each chapter there are copious supporting references to back up the textual material.

This book will appeal, in addition to radiologists, to neurologists, neurosurgeons and pathologists, who will find detailed descriptions of all of the pathologic processes likely to affect the head and the neck. Specifically, all chapters cover the normal roentgen anatomy and its variations, along with roentgenographic considerations, in order to make it possible for others to reproduce the results achieved by the authors. Very valuable are the chapters on infection of bone, including a discussion of all usual and unusual conditions, with reproductions of both the roentgenograms and the photographs of the pathologic specimens. For dental and maxillofacial surgeons an excellent chapter on the normal state and pathologic alterations of the jaws and teeth is presented. The relationship of dental conditions to systemic diseases, where roentgen manifestations may be present, is thoroughly discussed. The role of dental pathology in acting as focal infection is adequately covered. A comprehensive description of tumors involving the jaws is given.

Of particular interest to otorhinolaryngologists are the chapters on the nose and lacrimal passages, paranasal sinuses and the temporal bone. A good description of diseases of the nose and the lacrimal passageways is given. There is a complete and easily understandable coverage of the paranasal sinuses, detailing roentgenographic procedures along with a discussion of all possible disease conditions and their roentgen appearances. The techniques of examination of the mastoids, with excellent photographs of the desirable positions, are included. Full consideration of the advantages and disadvantages of the Mayer over the Owen modification of that position for best delineation of the mastoid antrum, the tympanic cavity and portions of the auditory canal wall is presented.

For the ophthalmologist there is adequate information on all practical methods devised for localization of foreign bodies in the eyeball,

along with a comprehensive discussion of the pathologic conditions of the eyes and orbits demonstrable by the roentgen method.

In the second volume intracranial tumors and other lesions of the brain and skull are dealt with, with detailed consideration of the cranial nerves and how they may be affected by tumors. There are excellent demonstrations of the various types of brain tumors. Many new illustrations which are well reproduced are of great aid in explaining this portion of the text. There are elaborate descriptions of roentgenographic techniques to be employed in order to achieve the best in roentgen demonstration. Clinical, pathologic and roentgen correlation has been done admirably, presenting the disease conditions as easily understood entities.

There is a very complete discussion of meningiomas both from the clinical and the roentgenologic points of view.

An outstanding chapter of the book deals with cerebral pneumography with lucid description of the technique, both operative and roentgenographic, to be employed. Detailed case histories with pathologic and roentgen findings make interesting and instructive reading.

There is an excellent analysis of the interpretative criteria given for encephalographic and ventriculographic studies, followed by discussion of both internal and external hydrocephalus, porencephalic lesions, herniations of the ventricles, and arachnoiditis, with illustrative cases.

A long chapter is devoted to the subject of cerebral angiography. A full discussion of the normal and pathologic roentgen anatomy, with emphasis laid on the subtle normal variations simulating pathologic alterations, is given. Many illustrations, including photographs of autopsy specimens are used to show the points of interest. There is an informative chart showing, in tabular form, the usual vascular displacements of arteries and veins observed in intracranial disease. This will prove most useful for quick reference to check various vascular patterns in different tumor locations. In this connection, it may be mentioned that most of the statements which might be considered controversial are indicated by numerical references to other authors, which makes identification easy.

The last five chapters of the book are devoted to the neck and its anatomic structures. Both morphologic and functional aspects of alterations are considered. Many anatomic cross-sections for correlation with laminagraphic examinations are included, enabling evaluation of the roentgen features based on anatomic comparison. Special attention is paid to the larynx and the associated soft tissues in the anterior superior portion of the neck. There is a complete discussion of the lesions in this region, particularly those involving the foramen magnum, the atlas, axis and the occipito-atlantoid articulation. Various types of tumors, occurring in the larynx, pharynx and esophagus, are considered, as well as the foreign bodies in the esophagus and trachea, and the various inflammatory changes which may show roentgen manifestations. Fractures and dislocations of the cervical vertebrae and lesions of the intevertebral disks are dealt with exhaustively.

Finally, lesions of the spinal cord, including tumors of both intramedullary and extramedullary type, as well as inflammatory changes, are described. The usefulness of myelography in the diagnosis of these conditions of the central nervous system is emphasized.

Altogether, this outstanding work gives meticulous attention to detail so that it serves as an encyclopedic reference book; at the same time, in its ensemble it stands out in bold relief from other works of the present, proving the immeasurable value of expertly wrought anatomic and roentgen anatomic illustrations.

Lewis E. Etter, M.D.

A Text-Book of X-Ray Diagnosis by British Authors in Four Volumes. Volume I. Edited by S. Cochrane Shanks, M.D., F.R.C.P., F.F.R., and Peter Kerley, C.V.O., C.B.E., M.D., F.R.C.P., F.F.R., D.M.R.E. Third edition. Cloth. Price, $18.00. Pp. 521, with 533 illustrations. W. B. Saunders Company, 218 West Washington Square, Philadelphia 5, Pennsylvania, 1957.

A textbook is usually defined as a standard work prepared for the study of a particular subject, as opposed to a reference book, which is meant primarily for consultation, rather than for consecutive reading. The editors of the British volumes of diagnostic roentgenology acknowledge in the preface to the third edition that they never intended to cover every nuance and refinement of this discipline. Their textbook was conceived to describe fundamental principles of interpretation, to maintain close

contact with commonly encountered clinical problems, and to reflect the best current practice of radiology. This editorial policy has met with considerable success, and their highly readable text has become a classic for English-speaking physicians: its first edition (1939/9) was reprinted five times (1941, 1943, 1944, 1945/5, 1948), the second (1950/1) one time (1952).

The first volume of the third edition, published in Great Britain by H. K. Lewis & Co. in October 1957, consists of the same five parts (divided in fourteen chapters) and index, found in the previous edition. A total of 94 pages have been added to the new volume, the major changes involving the first part, the Central Nervous System, rewritten by M. H. Jupe (who became a senior contributor following the death of Sir Hugh Cairns) in collaboration with D. W. C. Northfield. Part one has now 322 pages with 291 illustrations (as compared with 242 pages and 212 illustrations in 1950). Special emphasis has been laid on the various contrast examinations. For instance, the third chapter, Ventriculography and Encephalography, has 73 pages and 108 illustrations (as compared with 53 pages and 65 illustrations in the second edition), the fourth chapter, Cerebral Angiography (called "angeiography" until 1952) has 61 pages and 45 illustrations (as compared with 36 pages and 26 illustrations in the second edition), and the seventh chapter, the Spinal Cord, which includes myelography, has 33 pages and 35 illustrations (as compared with 15 pages and 10 illustrations in the second edition). Some outdated material has been weeded out, and even new bibliographic titles have been included. Meticulous attention has been paid to details, for instance Figure 41, which in the old edition showed only the middle fossa, has been rephotographed to visualize the entire section of the cranium, resulting in a more adequate demonstration of the pathology in the middle fossa. There are many pencil drawings, and these are often more helpful for identification of the significant details, than the reproductions.

The second part, the Teeth and Jaws, by H. M. Worth, has incurred only minor changes. Granuloma of the pulp is now called internal absorption (with granuloma of the pulp and pink spot in parenthesis), while the dignified term temporomandibular syndrome replaces the colloquial clicking of the joint, used in previous editions. Some of the reproductions in this part have been slightly enlarged. They are all in the negative, and are also otherwise of much better quality than in the remainder of the volume.

The third part, the Eye, by R. G. Reid, contains only a few new paragraphs and plates. Among the latter, two with orbital phleboliths, and one dacryocystogram, are negative reproductions, but all others, to the end of the volume, are in positive.

The fourth part, the Accessory Nasal Sinuses, and the fifth, the Ear and Temporal Bone, both by Sir Harold Graham-Hodgson, are also practically unchanged. In the fourth part there are two new plates, showing agger nasi cells, i.e., cells anterior to the frontonasal duct. All the reproductions in the fifth part have been made from new cuts, although in some cases the old roentgenograms may have been used.

The reproductions, with the exception of the above mentioned part two, are not of the highest quality. This printing, sponsored by the W. M. Saunders Company, was obviously intended for the American market, and perhaps an effort could have been made to have all the illustrations in negative. Otherwise the paper, the print, the general appearance exhibit a sophisticated taste. Above all, the educational value of the text, is beyond reproach. Even with the shortcomings of illustration, this volume is highly recommended to the graduate students in radiology, as well as to the practitioners in the specialties discussed in these chapters (neuro-surgery, E.E.N.T., etc.). Its perusal might constitute a pleasant refresher course for any radiologist. There is little doubt that this third edition will earn the same well-deserved success enjoyed by the previous printings.

E. R. N. Grigg, M.D.

Die gesunde und die kranke Wirbelsäule in Klinik. Pathologisch-anatomische Untersuchungen. By Geh. Med.-Rat. Prof. Dr. Dr. G. Schmorl; Für Röntgenkunde und Klinik by Prof. Dr. H. Junghanns, Oldenburg (Oldb.). Fourth edition. Cloth. Price, $20.50. Pp. 330, with 419 illustrations. Georg Thieme, Herdweg 63, Stuttgart, Germany, 1957.

This, the fourth edition of a standard work on the spine, first printed in 1932, has again been partially rewritten and enlarged. The anatomic and pathologic-anatomic studies of Schmorl remain original and they constitute the foundation upon which excellent chapters are contributed on the roentgen aspects of growth and normal structure of the spine, congenital anomalies, diseases of the bony structures of

the spine, diseases of intervertebral disks, and on the correlation of the anatomic, pathologic, roentgenologic and clinical aspects, in great detail and guided by many interesting ideas. Other chapters deal with spondylosis deformans, abnormal changes in curvatures of the spine, abnormalities of mobility of the spine, spondylolisthesis and the lumbosacral region.

While the clinical aspect receives constant attention, the book is not primarily a clinical study of diagnosis and therapy. Its main endeavor is to base the clinical and roentgen diagnosis on a thorough knowledge and understanding of normal and pathologic anatomy.

An index of the literature in this field occupies 42 pages, and is apparently complete up to 1955.

The reproduction of roentgenograms, photographs of the anatomic specimens and the entire make-up of the book are of the highest quality.

Hans W. Hefke, M.D.

The Treatment of Fractures. Volume II. By Lorenz Böhler, M.D., Director of the Accident Hospital, Vienna; Professor of Accident Surgery, University of Vienna. Translated from the Thirteenth German Edition by Otto Russe, M.D., Director of the Accident Hospital, Vienna, and R. G. B. Bjornson, M.D., Diplomate, American Board of Radiology. Cloth. Price, $17.50. Pp. 1,508, with 2,166 figures. Grune & Stratton, Inc., 381 Fourth Avenue, New York 16, New York, 1957.

The author follows the method of presentation used in the first volume. Injuries of the hip and entire femur are the subject matter of this volume. Diagnosis, the materials to be used, and their manner of utilization in the treatment of these injuries are presented in minute detail. He has included sections which cover all phases of the treatment of gunshot injuries of these areas.

As was stated in a review of the first volume, many American and British surgeons will not agree with the author's method in all instances. The style of this volume is more pedantic than provocative. It is easy to read and well illustrated. A list of errata which are numerical is included.

The two volumes present conclusions made by the author and his associates after consideration of a fantastically large volume of material. They justly deserve a place in the library of any surgeon interested in the treatment of trauma.

Frank P. Walsh, M.D.

Dental and Oral X-Ray Diagnosis. By A. C. W. Hutchinson, D.D.S., M.D.S., F.D.S., F.R.S.E., Professor of Dental Surgery, University of Edinburgh. Cloth. Price, $15.00. Pp. 524, with 946 illustrations. E. & S. Livingstone, Ltd., Edinburgh; The Williams & Wilkins Company, Mount Royal and Guilford Avenues, Baltimore 2, Maryland, 1954.

This is a book which can be recommended as an asset to the library of any person interested in oral diagnosis. In the preface, while stressing the importance of the roentgenographic examination, the author emphasizes the necessity for close cooperation between the pathologist, clinician and radiologist.

The first three chapters are devoted to development of the teeth, normal anatomic landmarks and their variations, and to the anomalies of the teeth. The next ten chapters are concerned with diseases of the teeth and peridental tissues and these are followed by a chapter on diseases causing inflammatory changes in the bone structure of the jaws. Then, the subjects of fractures of the facial bones, diseases of the maxillary sinus of interest in oral diagnosis, and disturbances of the temporomandibular articulations are presented. Developmental anomalies of the jaws are described and radiopaque shadows primarily in the soft tissues are mentioned. The next chapter, devoted to diseases of the salivary glands, is very interesting, as well as the two short chapters describing the roentgenographic evidence of diseases due to disturbances of the endocrine system and of nutrition. Then follow well illustrated chapters concerning osteodystrophies involving the jaws, cysts, odontogenic and nonodontogenic malformations, nonspecific granulomata, and tumors of epithelial and mesenchymal origin.

In addition to the roentgenographic descriptions, the author has included concise discussions of etiology, pathology and clinical features of the conditions presented. In a few instances, illustrations do not accompany the diseases described. However, in general, it must be stated that the book is well illustrated and the roentgenograms are very clearly reproduced.

Haven F. Doane, D.D.S.

Fortschritte der Kiefer- und Gesichts-Chirurgie; Ein Jahrbuch. Volume III. By Prof. Dr. Dr. K. Schuchardt, Direktor der Klinik und Poliklinik für Zahn-, Mund- und Kieferkrankheiten der Universität Hamburg und der Nordwestdeutschen Kieferklinik.

Cloth. Price, DM 96.—. Pp. 359, with 391 illustrations. Georg Thieme, Herdweg 63, Stuttgart, Germany, 1957.

This is the third yearbook, comprising the collected papers presented before the German Society of Maxillofacial Surgery, which met in Hamburg from the 26th to the 28th of July, 1956. The central theme of the meeting was "Malignant Tumors of the Maxillofacial Region."

The first portion of the book deals with the subject exhaustively from the point of view of pathology, biochemistry, surgery, maxillofacial surgery, radiology, dermatology, eye, ear, nose and throat diseases, plastic repair, and the oral and dental problems. The book portrays the many sided features in an admirable manner.

There are many illustrations throughout the 350 pages, with excellent photographs in black and white and numerous informative diagrams.

The use of roentgen therapy alone or in conjunction with surgery for treatment, the subjects of immunity to carcinoma and resistance of tumors, and many other topics are discussed in detail. An interesting feature of the book is a summary in German, English and Spanish at the end of each chapter, adding much to its readability and to its usefulness as a ready reference.

The printing is beautiful, with sharp, clear and distinct type on highly glossy paper of very convenient size, and does credit to the Publishing House of Georg Thieme of Stuttgart, Germany. The editor should be congratulated for being able to collect such a large series of papers from so many individuals and present them in such readable and understandable form. This in itself is a great achievement. However, there are also several interesting chapters on the use of angiography for the diagnosis of the tumors of this region. This somewhat new approach is to be commended as long as it is used with judgment and care; it has possibilities of contributing considerably to the solution of many vexing problems.

Incidentally, a convenient little bookmarker is attached, a thing the reviewer has not seen for many a year and which is a nicety the reader often enjoys and finds useful.

All in all, this is an excellent presentation which can be read with pleasure and profit by those interested in maxillofacial surgery and informatively by those practicing in other specialties.

Eugene A. Ossius, M.D.

BOOKS RECEIVED

Duodénum et Pancréas. By Guy Albot, Médecin de l'Hôtel-Dieu; and F. Poilleux, Chirurgien de l'Hôpital Corentin-Celton. Paper. Price, 3,500 fr. Pp. 296, with 126 illustrations. Masson et Cie, Éditeurs, 120 boulevard Saint-Germain, Paris 6e, France, 1957.

The Alimentary Tract of the Ruminant. By David Benzie, and A. T. Phillipson, M.A., Ph.D., M.R.C.V.S., F.R.S.E., The Rowett Research Institute, Bucksburn, Aberdeenshire. Cloth. Price, $5.50. Pp. 24, plus 54 plates. Charles C Thomas, Publisher, 301 East Lawrence Avenue, Springfield, Illinois, 1958.

Traitement chirurgical de la Recto-côlite Ulcéro-Hémorragique. By E. Delannoy, Professeur de Clinique chirurgicale à la Faculté de Médecine de Lille; and M. Martinot, Moniteur de Clinique chirurgicale à la Faculté de Médecine de Lille. Paper. Price, 1,400 fr. Pp. 120, with 26 illustrations. Masson Cie, Éditeurs, 120 boulevard Saint-Germain, Paris 6e, France, 1957.

La Broncografia nella Fisiopatologia Bronchiale. By Dario Gandini and Giovanni Juliani. Cardboard. Price, L. 4,000. Pp. 200, with 141 illustrations. Edizioni Minerva Medica, Corso Bramante 83–85, Torino, Italy, 1957.

A Study of the In Vitro and In Vivo Effects of a Synthetic Heparin-Like Anticoagulant: Dextran Sulphate. The Scandinavian Journal of Clinical & Laboratory Investigation, Supplement 29. By Peter Hjort and Helge Stormorken; with Capillary Microscopy by Oscar Gilje. Paper. Pp. 86, with 28 figures and 18 tables. The Scandinavian Journal of Clinical & Laboratory Investigation, Medical Department A, Rikshospitalet, Oslo, Norway, 1957.

Symposium on Antibodies: Their Production and Mechanism of Action. Paper. Free of charge to scientific investigators. Pp. 361, with figures and tables. Oak Ridge National Laboratory, Post Office Box Y, Oak Ridge, Tennessee, 1957.

Tumors of the Soft Somatic Tissues. By George T. Pack, M.D., LL.D., F.A.C.S., Attending Surgeon, Memorial Center for Cancer and Allied Diseases; Associate Professor of Clinical Surgery, Cornell University Medical College; Surgeon, Pack Medical Group, New York; and Irving M. Ariel, M.D., F.A.C.S., Associate Clinical Professor of Surgery and Associate Attending Surgeon, New York Medical College, Flower and Fifth Avenue Hospitals; Surgeon, Pack Medical Group, New York. Cloth. Price, $3.00. Pp. 820, with 652 illustrations. Paul B. Hoeber, Inc., 49 East 33rd Street, New York 16, New York, 1958.

Radium-Isodosen; Die Radiumdosierung in "r." By Dr. Med. August Verhagen, Oberarzt an der Städt. Frauenklinik Essen (Ruhr). Paper. Price. DM 19.50. Pp. 96, with 34 illustrations. Georg Thieme, Herdweg 63, Stuttgart, Germany, 1958.

Vol. 79, No. 4

ABSTRACTS OF RADIOLOGICAL LITERATURE

Department Editor: T. Leucutia, M.D., Harper Hospital, Detroit 1, Michigan

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ROENTGEN DIAGNOSIS

Head

Frey, K. W., and Mündnich, K. Schichtaufnahmen des Felsenbeines mit polyzyklischer Verwischung bei angeborenen Ohrmissbildungen. (Laminagraphy of the petrous bone with polycyclic tube movement in congenital abnormalities of the ear.) *Fortschr. a. d. Geb. d. Röntgenstrahlen u. d. Nuklearmedizin*, August, 1957, *87*, 164–176. (Address: K. W. Frey, Institut und Poliklinik für Physikalische Therapie und Röntgenologie der Universität München, Ziemssenstr. 1 a., München, Germany.)

While congenital ear abnormalities are rare, recent development of operations for improvement of hearing in these patients have increased interest in roentgen examination of these cases. Besides routine roentgenography, the authors use anteroposterior and lateral laminagrams of the petrous bone, axial laminagrams perpendicular to the long axis of the petrous pyramid and laminagrams with axial (presumably submento-vertical) position of the skull. Occasionally laminagrams were also obtained in Chaussé III and Stenvers' projections. Two diaphragms, one at 12 cm. distance from the anode of 0.7 cm. diameter and a second at 36 cm. with 2 cm. diameter were used. Details of the roentgenographic technique were published by Frey.

Three main groups of congenital abnormalities are described by the authors: (1) A large cavum tympani, attic and antrum and usually very good pneumatization; (2) a small cavum tympani and narrow attic and bony or soft tissue adhesions in the middle ear. The antrum can be large with pneumatization of the mastoid process or it can be small or missing; and (3) small middle ear spaces combined with hard, sclerosed bony walls and often accompanied by other facial malformations.

The article contains numerous reproductions of laminagrams with line drawings explaining the findings and a considerable number of references.—*Karol E. Matzinger, M.D.*

Capps, F. C. W. Tumours of the glomus jugulare or tympanic body. *J. Fac. Radiologists*, July, 1957, *8*, 312–324. (From: St. Bartholomew's Hospital, London, England.)

Since the description by Guild in 1941 of a body in the adventitia of the jugular bulb or on branches of the 9th or 10th nerves in the middle ear which was analogous to the carotid body, and Rosenwasser's paper concerning a "carotid-body-like" tumor in the middle ear, the author believes it has become recognized that most of such tumors arise from the glomus jugulare of Guild,—also known as the tympanic body. These lesions occur most often in middle-aged females. The gross appearance is not specific—the usual impression, when found casually in the external auditory meatus being that of aural polyps, secondary to middle ear disease. Histologically they closely resemble the architecture of the normal body as described by Guild and of the carotid body. They are made up of nests of "chief" or epithelioid cells, of fairly uniform size and surrounded by vascular stroma. Metastasis is uncommon but has been reported occasionally to lymph nodes, liver, lung and spleen.

Clinically these lesions appear as very vascular tumors of the external auditory meatus, middle ear and surrounding structures. They are observed in two groups clinically. In the first group they arise in the tympanic cavity and pass through the ear drum to form meatal polypoid tumors. They are usually devoid of nerve involvement and may give rise to pulsing tinnitus, deafness with pain, and vertigo. In the second group they arise in the neighborhood of the jugular bulb. They spread on the under surface of the petrous bone, sometimes with bone destruction and involve structures in the jugular foramen (9th, 10th and 11th cranial nerves), in the anterior condylar foramen (12th cranial nerve), and may spread by way of the foramen lacerum anterior to the middle fossa of the skull (5th and 6th nerves). Sooner or later they invade the tympanic floor and involve the 7th cranial nerve and the labyrinth.

The author presents the case histories of 3 patients in Group 1,—those with meatal polypoid tumors originating in the tympanic cavity, and of 4 patients in Group 2,—those in whom the tumors arise in the neighborhood of the jugular bulb.

In diagnosis, roentgenography of the petrous bone gives valuable information of the degree of local spread, and laminagrams may show bony defects not evident on routine films. The most common error is to make a diagnosis of acute otitis media in patients with a bulging red drum membrane. Polypoid tumors in the auditory meatus are often mistaken for granulomatous polyps seen secondary to chronic otitis media.

Radiation therapy gives quite lasting improvement, but it has little effect on the analogous carotid-body tumors. Shrinkage of the tumor is probably brought about by thrombosis of the vessels. The author believes that better results are obtained when roentgen therapy is given after removal of the polyps, or after performance of a tympano-mastoidectomy. In planning the therapy the entire volume of tissue to be irradiated is determined. Using isodose charts, fields are planned to cover this region and to deliver a tissue dose of 4,000 to 6,000 r in six weeks. In suitable cases (the first or tympanic group) operation may give long lasting cure and here one can safely withhold radiation therapy until evidence of recurrence is noted. The author believes that surgical removal by radical tympano-mastoidectomy should be limited to this first group of tumors.—*Samuel G. Henderson, M.D.*

Harper, R. A. Kemp. Glomus jugulare tumours of the temporal bone. *J. Fac. Radiologists*, July, 1957, *8*, 325–334. (From: Diagnostic Radiology Department, St. Bartholomew's Hospital, London, E.C.1, England.)

The roentgenologic features vary considerably according to the extent of the glomus jugulare tumor. Sclerosis of the mastoid and petrous regions may be the first sign, and may simulate an old infected mastoid. Laminagraphy is important to demonstrate the early degree of destruction of the petrous bone as the tumor spreads from the region of the jugular bulb. Later, the middle ear and labyrinth tend to be destroyed. As the bony structure crumbles away, large defects appear in the petrous bone. In the advanced stage of the tumor, the whole of the petrous area and much of the mastoid and squamous portions of the temporal bone may disappear.

The jugular foramen, seen in submento-vertical views, may be enlarged early, and later in this view the destruction may include the carotid canal and the petrous apex. In the occipital projection one may see erosion of the articular process of the occipital bone and invasion of the temporo-mandibular joint on its medial aspect. Due to the great vascularity of the tumor the groove for the emissary vein of the mastoid process may be much enlarged. With destruction of the overlying petrous bone, roentgenograms in the submento-vertical view may show the vertebral canal in the first cervical vertebra more clearly, and enlargement of the first cervical vertebral foramen may be evident. Cerebral angiography will probably not be necessary in the majority of cases, but it may be helpful in the early stages, especially in differential diagnosis because of the great increase in vascularity of the mastoid and petrous bone.

The author describes the most useful views for demonstrating these tumors: (1) the lateral oblique or Schüller view; (2) the occipital view, tilting the tube 30 degrees toward the feet; (3) the submento-vertical view, 20 degrees short of the full axial projection, so that the jugular foramen is projected just below the inferior border of the mandible; (4) the same view with Chaussé's variation of an additional lateral tilt of the head toward the affected side; (5) laminagraphy in the anteroposterior view; and (6) in doubtful cases, cerebral angiography.

Six case reports, 5 females and 1 male, are given.

Differential diagnosis includes carcinoma of the middle ear, cholesteatoma, nasopharyngeal tumor, acoustic neuroma and rarely chordomas, and metastatic lesions in the mastoid area.

In carcinoma of the middle ear, while there is extensive destruction of the mastoid area, the destruction tends to spread more laterally than medially, so that the destruction of the petrous apex is not so great in carcinoma and the inferior portion of the petrous bone is less apt to be destroyed. In cholesteatoma, the lytic area is usually well defined and it does not extend so far medially or inferiorly as in the case of glomus tumor. In nasopharyngeal tumor, there is the presence of a large mass in the nasopharyngeal space and bilateral rather than unilateral bone destruction. Acoustic neuroma may cause extensive petrous destruction, but it is usually well defined and does not involve the inferior portion of the petrous bone, nor is the moth-eaten erosion of the remainder of the mastoid area seen in acoustic neuroma.—*Samuel G. Henderson, M.D.*

Williams, I. G. Radiotherapy of tumours of the glomus jugulare. *J. Fac. Radiologists*, July, 1957, *8*, 335–338. (From: Radiotherapy Department, St. Bartholomew's Hospital, London, England.)

The reports on therapy of tumors of the glomus jugulare up to 1949 were few, but all were unanimous in classifying the lesion as radioresistant.

The following factors were taken into consideration in planning the treatment of this tumor in patients with distressing clinical symptoms and considered nonoperable:

1. The clinical behavior of the tumor. The growth arises in the glomus jugulare, but may also occur in the first part of the tympanic canal and along the auricular branch of the vagus, as far forward as the facial canal. The tumor is slow growing but progressive, very vascular, locally invasive, but nonmetastasizing, and may involve the regional cranial nerves. It produces deafness, tinnitus, upset of balance, and cranial nerve neurologic manifestations.
2. Because of the relatively benign course the therapy should not endanger the patient by infection or by radiation brain necrosis.
3. The close resemblance between glomus jugulare tumor and carotid body tumor and the absence of clinical regression in the latter tumor after high radiation dosage indicate that high dosage would be necessary to alter the course of glomus jugulare tumor.
4. The vascularity of the tumor might enable one to produce an endarteritis and cut off the tumor's blood supply.
5. Since the tumor is surrounded by bone, very high voltage radiation should be more effective than low voltage radiation because the dose at the tumor is much nearer the predicted dose.

The author treated 10 patients with 1 mev., half-value layer of 10 mm. Cu, focus skin distance of 100 cm.; and two patients with 250 kv., half-value layer of 1.8 mm. Cu. Size of fields varied from 5×5 cm. to 9×16 cm. Two oblique fields were used anteriorly and posteriorly in most of the patients. In some, in order to obtain a wedge filter effect and adjust the isodose, narrower supplementary fields were employed. Dosage was:—4,000 r (at level of tumor) in twenty-eight days in 6 cases, 5,000 r in thirty to forty days in 3 cases, 2,275 r in fifteen days in 1 case

(treatment terminated because of a severe B. pyocyaneus infection). In the 2 patients treated with 250 kv., 3,500 r was given in four weeks.

Results. Shrinkage of tumor in all cases. The patient who developed B. pyocyaneus infection has minimal symptoms two years later, with residual tumor. One patient was operated on and persistence of tumor was found four years after therapy. Symptoms have been relieved in all cases, except for deafness. In 3 patients with severe nerve pareses good recovery has resulted. The fact that in 2 patients, one treated to 5,000 r, and the other to 4,000 r, the tumor did recur, indicates that it is difficult to be certain that the tumor is completely destroyed.—*Samuel G. Henderson, M.D.*

Kindler, W. Röntgenologische Untersuchungen eines künstlich deformierten Schädels aus der Völkerwanderungszeit. (Roentgenologic examination of an artificially deformed skull from the peoples' migration period.) *Fortschr. a. d. Geb. d. Röntgenstrahlen u. d. Nuklearmedizin*, August, 1957, *87*, 185–190. (Address: Vosstr. 5, Heidelberg, Germany.)

Artificially deformed skulls of human skeletons belonging to the pre-Columbian period were discovered on the American continent; the deformity consisting in an elongation and narrowing of the parieto-occipital segment of the skull. A similarly deformed skull from the sixth or seventh century A.D. was discovered in Germany recently and a photograph, as well as 5 roentgenograms are reproduced in this article. This skull deformity is also found in some African Negro tribes, where a special cap is used to produce an elongation of the neurocranium in female infants.

This article, containing 5 references in German, should be of value to those interested in the use of roentgenography in anthropology.—*Karol E. Matzinger, M.D.*

Neck and Chest

Hausser, R., and Grimminger, A. Über die Luftzystenerkrankung der Lunge; (Vanishing lung nach Burke, progressive Lungendystrophie nach Heilmeyer und Schmid). (Cystic disease of the lung; [vanishing lung of Burke, progressive pulmonary dystrophy of Heilmeyer and Schmid]. *Fortschr. a. d. Geb. d. Röntgenstrahlen u. d. Nuklearmedizin*, Sept., 1957, *87*, 283–307. (Address: R. Hausser, Heilstätte des Württ. Landesfürsorgeverbandes, Gundelsheim [Neckar], Germany.)

Pulmonary cysts may be classified along pathologic-anatomic lines as follows: (1) congenital or acquired; (2) bronchial, alveolar, interstitial, or hamartomatous; (3) fluid- or air-filled; (4) inflammatory or degenerative, rarely traumatic; and (5) single or multiple. For clinical purposes, the cysts may be divided into two main groups: (A) Alveolar cysts; solitary (pneumatocele), or cystic emphysema; (B) bronchial cysts (air- or fluid-containing); solitary (pneumocyst), or multiple (cystic bronchiectasis). The two main groups may be further subdivided into expandable or nonexpandable and diffuse or localized cysts. Since it is often difficult to differentiate between these two groups clinically, unless the case history is known from the onset, the authors prefer the term "air cyst."

Cysts may develop because of a lack or loss of elasticity of the lung structures; as an aftermath of infection; and, especially, a combination of both. An air-filled cyst cannot be considered a congenital manifestation because air in the lungs is a postnatal occurrence. "Congenital lobar emphysema" and "congenital cystic lung disease" are therefore considered misnomers. The congenital or primary defect, however, is partial or complete failure of development of elastic tissues. This accounts for the frequency of spontaneous pneumothorax in infants. Postnatal disorders, which contribute to cyst formation, include failure of degeneration of mucosal folds, anomalies of the bony thorax, bronchospasm, etc.

A case is presented in which the development of a cyst was traced from a nonspecific infiltrate, characterized as a small zone of atelectasis, within which central necrosis occurred and a cherry-sized, thin-walled cyst appeared. After three to four weeks, the cyst disappeared and was replaced by scar tissue. During the stage of infiltration, a functional bronchospasm and a kinking of the bronchus combine to act as a valve mechanism and the cyst increases in size. This sequence is possible only if the tissue adjacent to the cyst is diseased, so that collateral ventilation cannot take place through the alveolar pores. The cyst disappears when the bronchus remains open and the entrapped air is absorbed. In this case, the presence of a valve mechanism had caused the cyst to enlarge. Whenever there is a broad communication with a bronchus, a cyst will not change in size.

Cysts may be asymptomatic until they cause compression or atelectasis with subsequent infection. A subpleural cyst may rupture and produce a spontaneous pneumothorax if no pleural adhesions are present. If the cyst wall acts as a valve mechanism, a tension pneumothorax may be established and cause massive lung collapse. Excision of the cyst becomes mandatory if the situation persists and causes infection. When a small zone of parenchymal tissue separates the cyst from the pleura, the area eventually becomes atelectatic as the cyst enlarges. The atelectatic zone unites with the pleura to form a resistant fibrous wall, which, protects against a rupture and spontaneous pneumothorax. Giant cysts can occur only if the pleural space is free. The term, "intrapulmonary pneumothorax" refers to giant cysts, which may be so large as to involve an entire

hemithorax. Such cysts may develop so slowly as to produce no symptoms. Cysts located near the hilus cannot reach giant size because the hilar bronchi and vessels act as a resistance to expansion.

The diagnosis of a cyst, in the absence of clinical signs, may be an accidental finding on roentgenographic examination. If the cyst is round, it will be recognized by its thin wall, the increased radiolucency, and the diminution or absence of blood vessels. Multiple, small cysts may go unrecognized because their borders are ill-defined. However, their presence may be suspected if the hilus is displaced and if the vessels and bronchi are crowded together. Laminagraphy and bronchography are also helpful in diagnosis, especially in differentiation from pneumothorax.

The treatment of cysts is surgical removal, especially if they cause compression symptoms. If pleural symphysis exists, puncture, or Monaldi drainage may be adequate. However in older patients, drainage may have to be permanent and infection may ensue requiring subsequent cystectomy. If the pleural space is free, drainage should not be tried unless preparation for immediate thoracotomy is made in advance because of the danger of a tension pneumothorax. If the cyst is centrally situated and reaches the hilus, lobectomy is required.

The terms air cyst, vanishing lung (Burke), and pulmonary dystrophy (Heilmeyer and Schmid) are synonymous; although the latter diagnosis is considered a separate entity by Heilmeyer and Schmid, who believe the primary defect to be a disease of the pulmonary and bronchial arteries followed by a total disappearance of the lung parenchyma. The authors of this article cannot accept this concept, since they have seen complete restoration of function and no signs of atrophy in portions of lungs which have been compressed or atelectatic for long periods of time. The authors have aligned themselves with those who support the mechanical concept of cystic disease.—*J. Zausner, M.D.*

RADIOISOTOPES

Coliez, R. T., Tabiana, M., and Alagille, D. Traitement par le phosphore radioactif des thrombocythémies et des polyglobulies. (Treatment of thrombocythemias and polycythemias with radioactive phosphorus) *J. de radiol., d'électrol. et de méd. nucléaire*, May–June, 1957, *38*, 405–410. (From: Laboratorie des Isotopes de l'Institut Gustave-Roussy, and Centre de Traitment des Tumeurs de l'Hôpital Necker, Paris, France.)

The authors report on 8 cases of thrombocythemia treated with radioactive phosphorus. In all cases, doses of 1 mc of P^{32} per 10 kg. of body weight have normalized the platelet count in four to six weeks without serious anemia or leukopenia. The clinical manifestations, including neurologic signs and hemorrhages, disappeared when the platelet count returned to normal; also the coagulation disorders have regressed during the period of remission.

One hundred and twenty-five cases of polycythemia have also been treated with P^{32}. In all but one, this treatment has provided excellent results. The authors feel that the radiosensitivity for P^{32} is the same whatever the disease duration and the red blood count may be.—*Maurice Dufresne, M.D.*

Bercy, A. Calcul de la dose d'irradiation reçue aux ovaires lors d'un traitement ou d'un test par l'iode radio-actif I^{131}. (Calculation of the dose of irradiation received by the ovaries during a therapeutic use or diagnostic test of radioactive iodine I^{131}.) *J. belge de radiol.*, 1957, *40*, No. 3, 333–345. (From: Service de Radiothérapie de l'Université, Liège, Belgium.)

The author's goal was to determine to what degree treatment of the hyperthyroid female with radioactive iodine could be harmful to the ovaries.

There were 2 ways to investigate this problem: (1) by vaginal introduction of an ionization chamber; and (2) by approximate calculation. For practical reasons the author chose the second method. On treatment of hyperthyroidism with radioactive iodine I^{131}, the radiation received by the ovaries is emitted by the whole body, though unequally through its various parts.

Four different sources of irradiation can be distinguished: (1) The thyroid gland—its influence upon the ovaries can be neglected due to the distance and the tissue thickness which separates the thyroid from the ovaries; (2) the urinary bladder—its action lasts about twelve hours. During this time the amount of radioisotope in the bladder, the shape of the bladder, the distance from the ovaries change continuously. By using an artificial bladder, the author estimated the dose of gamma rays to be 206 mr and the dose of beta-rays as none; (3) the colon—in a case of constipation it can emit a dose of 48 mr to the right ovary; and (4) the whole body—the circulating radioactive iodine contributes (a) an estimated beta-ray dose of 0.8 rep and (b) an estimated gamma-ray dose of 0.8 r.

Altogether a person partaking 10 mc of radioactive iodine (50 per cent of it being fixed in the thyroid with a half life of five days) receives to the ovaries a dose corresponding to 1 r and 0.8 rep and a total dose of 1.64 rad.

This dose of radiation to the ovary is one-third to one-fourth of the dose received on roentgen examination of the intestines, and is equal to the dose received on roentgen examination of the lumbosacral spine.

In the diagnostic test of the thyroid the ovary re-

ceives only 1/100th of this amount.—*Hans Lewin, M.D.*

CHEMOTHERAPY

Shapiro, Daniel M. Quantitative biochemical differences as a basis for cancer chemotherapy. *Radiology*, August, 1957, *69*, 188–193. (Address: University of Miami, Miami, Fla.)

Cancer tissue possesses the identical enzymes or other biochemical components known to be present in normal tissues. The difference between the cancer cell and the normal cell is ascribed to alterations in relative amounts of activities of the individual biochemical constituents. These differences between normal cells and tumor cells render the neoplastic cell vulnerable to appropriate chemotherapy.

Preliminary studies of vitamin B_6 metabolism revealed a much lower concentration of vitamin B_6 in mammary carcinoma 755 grown in mice, than in normal tissues. The B_6 antagonist, desoxypyridoxine, was tested and found to possess a weak but definite carcinostatic effect against this tumor. In an effort to obtain greater activity a number of antimetabolites were given simultaneously.

It was demonstrated that tumor regression resulted from simultaneous administration of compounds at levels which individually were incapable of producing results. Since radiation therapy probably produces its effects by simultaneous damage of many enzymes, the author combined these two modalities of treatment. The results of the combination of radiation therapy and multi-combination chemotherapy are reported in a subsequent paper.—*Arno W. Sommer, M.D.*

Kligerman, Morton M., and Shapiro, Daniel M. Augmentation of radiotherapeutic effect by cancer chemotherapy. *Radiology*, August, 1957, *69*, 194–200. (Address: M. M. Kligerman, 622 West 168th St., New York 32, N. Y.)

Various biochemical means have been used to sensitize cancer cells to ionizing radiations or to augment the effect of irradiation. Synthetic vitamin K, 8-azaguanine, and increased oxygen concentration each have been found to enhance the effect of radiation in tumor cells.

The authors have conducted experiments to determine the effect of radiation therapy and various combinations of chemical antimetabolites on mammary adenocarcinoma 755 in mice. The chemicals included 8-azaguanine, testosterone propionate, desoxypyridoxine, and 6-aminonicotinamide. All tumors treated by chemicals alone showed prompt regression but there was eventual escape of all tumors. Regression of those treated with roentgen rays alone was not consistent in all instances; it occurred more slowly, but in those that did respond, the period of regression was more prolonged. Tumors receiving combined radiation therapy and multi-combination chemotherapy showed earlier, greater and more prolonged response.

Some of the compounds used may be too toxic for human administration. Nevertheless, it is felt that the results have established principles for further attempts at cancer therapy. Radiation therapy and multi-combination chemotherapy both appear to be in part anti-enzymic in action. The combination shows promise of more effective control of human neoplastic disease.—*Arno W. Sommer, M.D.*

Loken, Merle K. Porphyrins as modifiers of the effects of roentgen rays. *Radiology*, August, 1957, *69*, 201–203. (Address: 412 Delaware St., S. E., Minneapolis 14, Minn.)

The author presents a summary of the investigations on chemicals as modifiers of the effects of roentgen rays on various biologic systems. Studies were undertaken to determine the factors in the *in vivo* and *in vitro* radiosensitivity of the systems treated with "hematoporphyrin."

Porphyrins and heme compounds have been reported to have many effects which are similar to the effects produced by ionizing radiations. Among these are protein denaturation, oxidative reactions, release of histamine or histamine-like compounds, increase in cell permeability, and carcinogenesis. Hematoporphyrin is composed of many different porphyrins, which vary greatly in their propensity for localizing in tumor tissues and various other tissues. The purest fractions so far available localize poorly and within one to two days after injection show little red fluorescence in the transplanted mouse mammary carcinoma. Some of the minor porphyrin contaminants show better localization in tumor tissue. There is also considerable variation in the toxic effects of different preparations.

Because of these reported results, the author is conducting studies with various fractions and combinations of fractions. Fifty-one patients with inoperable tumors were treated with combined porphyrin roentgen therapy. The response of one-third or more of the treated neoplasms was appreciably greater than expected. Further study, therefore, seems to be warranted with this and other porphyrin preparations.

Results have not been encouraging following the use of hematoporphyrin combined with nitrogen mustard, compound F, or myleran in rats with transplanted Walker carcinoma or in mice with transplanted mammary carcinoma. Studies of the combination of roentgen therapy and the copper complex of hematoporphyrin appear most encouraging to date. Findings with folic acid analogues combined with roentgen therapy are also quite encouraging. Studies with these and other chemicals are being continued.—*Arno W. Sommer, M.D.*

Thomas, Sydney F. Combined therapy: Radiation and chemicals. *Radiology*, August, 1957, *69*, 204–208. (Address: Palo Alto Medical Clinic, Palo Alto, Calif.)

The author has used a combination of irradiation and one or more chemicals in the treatment of over 100 patients with malignancy. In 8 patients, CB 1348, myleran, 6 mercaptopurine, aminopterin, and testosterone were used in combination with irradiation. These chemicals were administered toward the end of the course of roentgen therapy in each case. These studies were completed too recently to permit definite conclusions. Others have reported the use of substances such as urethane, testosterone, and hematoporphyrins in combination with irradiation.

One of the outstanding palliative uses of chemicals in conjunction with irradiation is in the patient with intractable pelvic pain from persistent carcinoma. Here the author has given nitrogen mustard locally to the pelvis by the intra-aortic route. Over 60 such injections have been done. The injection of 20 mg. of nitrogen mustard is made through a very fine polyethylene catheter inserted into the femoral artery and advanced under fluoroscopic control to a point below the renal arteries. Tourniquets are placed on both legs before injection and are not released until 5 minutes after injection. Relief of pain takes place within a few minutes to an hour or two following the injection. Shortly after this treatment a course of irradiation is given to the pelvic region. Besides the relief of pain there appears to be pathologically demonstrable evidence of an enhanced destructive effect on malignant tissue.

Unpleasant side effects have followed the use of some of these substances, but they have, for the most part, subsided promptly. It is predicted that in the future a poly-active chemical or chemicals will be used with irradiation to ablate abnormal growth processes.—*Arno W. Sommer, M.D.*

MISCELLANEOUS

Mauderli, W. Dosimetrie von Röntgen- und Gammastrahlen mittels photographischer Filme. 1. Teil: Physikalische Grundlagen; 2. Teil: Anwendung auf die Filmdosimetrie. (Dosimetry of roentgen and gamma rays by means of photographic films. Part 1: Physical fundamentals; Part 2: Use of film dosimetry.) *Fortschr. a. d. Geb. d. Röntgenstrahlen u. d. Nuklearmedizin*, May and June, 1957, *86*, 634–642 and 784–794. (Address: 1518 S. Fillmore, Little Rock, Ark.)

Dosage methods in radiology are most important, both for the measurement of therapeutic doses and the determination of protection doses to which persons in their work may possibly be exposed. The energy absorbed in the irradiated object is essentially responsible for the biologic effect produced. A dosage instrument must therefore fulfill the following conditions: It must measure the radiation for the entire energy range used in medicine in such a manner that a definite relationship exists between the measured value and the biologic effect. For a wide range of wavelengths this also signifies that the measured value always is a measure of the absorbed energy. From a medical point of view, this would mean that the measured value is independent of the wavelength. Usually a detector is considered independent of the wavelength if its measured values are the same as or parallel to those of an air ionization chamber. The author describes the study of the dependence of photographic films upon the wavelengths of the radiations used.

Dosage measurements with photographic films are mainly used in surveying persons who are exposed to radiations. Compared to ionization chambers, films have the advantage of being less sensitive to humidity and dust. They have a negligible zero effect and therefore lend themselves to long times of exposure and they represent a permanent record of the exposure received. Disadvantages are their smaller accuracy of measurement and their large dependence upon the wavelength.

In a thorough theoretic and experimental study, the author examines the relationship between the darkening of silver bromide emulsions by roentgen rays and the radiation energy absorbed within them. Absorbed energies were calculated with Gray's formula from ionization measurements made with a condenser r-meter. It was found that the photographic density depends not only upon the absorbed energy but also upon the mean ion density, whereby gelatin content and grain size play an important role. Even experiments with extremely thin silver bromide emulsions (of the order of 2.2 mg./cm.^{2} gelatin and 1.7 mg./cm.^{2} silver bromide) did not show a great decrease in the dependence of absorbed energy upon the wavelength, due to the effect of the ion density. Attempts to compensate for the wavelength dependence by filtering the roentgen-ray beam with special cadmium, selenium and silver bromide filters showed that for certain ranges of wavelength a compensation could be accomplished but not one filter alone could produce compensation for the whole range of wavelengths used in medicine. A detailed discussion follows of errors and obtainable accuracies in determining dosages with Ilford P.M. 1 (gamma rays) and P.M. 3 (soft roentgen rays) films, such as calibration of standard films with ionization chambers, inhomogeneity of film emulsions, film development, measurement of film density, dependence upon wavelength and fading effect of films. Measurement of film density by means of the optical Goldberg densograph of Zeiss-Ikon and a photoelectric densitometer of the author's design is discussed in detail and an analysis of the H.&D. density curves obtained is given.—*Otto Glasser, Ph.D.*

Hubbell, Harry H., Jr., Johnson, Raymond M., and Birkoff, Robert D. Design and calibration of pocket personnel dosimeters for beta radiation. *Radiology*, August, 1957, *69*, 268–273. (Address: H. H. Hubbell, Jr., Post Office Box P, Oak Ridge, Tenn.)

The authors describe a pocket dosimeter suitable for measurement of beta radiation. It is designed to meet the need for satisfactory techniques for personnel monitoring to avoid overexposures to beta radiation. The chamber is a modification of the standard Victoreen pencil ionizing chamber and can be charged and read in the same Minometer. Several models with different perforated protecting walls have been made. Chambers with perforated aluminum or magnesium walls 0.010 inch thick proved quite successful. The dosimeter was calibrated by placing it in a cavity having 1 mg./cm.^{2} walls within a tank containing dilute water solutions of several beta-emitting isotopes. The dose rate was calculated from the known beta-energy spectrum and the measured specific gravity of the solutions, according to the Bragg-Gray principle.

The perfected personnel beta dosimeter permits determination of either combined beta- and gamma-ray dose, or of beta-ray dose alone if a standard chamber is also worn and its reading subtracted from that of the beta chamber.—*Arno W. Sommer, M.D.*

Nahon, Joseph R. Back-scatter from soft tissue and bone. *Radiology*, August, 1957, *69*, 255–258. (Address: South Center St. and Tremont Ave., East Orange, N. J.)

It has been the general impression of most radiotherapists that backscatter from bone is greater than that from soft tissue. Verification of this impression was undertaken with the comparison of bone equivalent and soft tissue equivalent phantoms.

These studies showed that backscatter from bone is less than that from soft tissue. Increase in the field size increased this difference. The peak percentage backscatter from bone was found to be associated with a higher half value layer than the peak percentage backscatter from soft tissues.—*Donald N. Dysart, M.D.*

Maisin, J., Maldague, P., Dunjic, A., and Maisin, H. Syndromes mortels et effets tardifs des irradiations totales et subtotales chez le rat. (Lethal syndromes and late effects of total and subtotal irradiation in rats.) *J. belge de radiol.*, 1957, *40*, No. 3, 346–398. (From: Laboratoires de Recherches pour la Protection de la Population Civile annexés au Département de Radiologie de l'Institut du Cancer de l'Université Catholique de Louvain, Louvain, Belgium.)

This article is too extensive for detailed review. The authors reiterate the various causes of death in rats after total and subtotal irradiation which they had reported previously in several articles. In this article they describe specific fatal syndromes occurring within the period of seventy days after irradiation, namely: the medullar, intestinal, oropharyngeal, esophageal and pulmonary syndromes. The authors investigated the survival rate of the animals still alive thirty days after irradiation. They found a shortening of life also in these animals.

Various protective and therapeutic agents which improved the survival rate up to the thirtieth day failed to improve the rate thereafter. Even local irradiation of head, thorax and abdomen shortened life. The authors conclude that perhaps shortening of life after total body irradiation is due to the summation of damages inflicted upon single organs, and that there exists a threshold dose of irradiation for each organ.

Lethal gross pathology and the incidence of cancer in their experimental series are described. Most of the neoplasms of the nonirradiated control animals were leukosarcomas, whereas the irradiated animals presented carcinomas of very different types. The number of neoplasms of the irradiated rats was double that of the control animals. Neoplasms never developed in parts of the body which were protected by a lead shield during irradiation. This was also the only protective measure which improved the survival rate after the thirtieth day. The authors followed the late results of irradiation upon a generation of rats which were irradiated with roentgen rays in utero and were protected by MEA (cysteamine) injection of the mother rat before the irradiation. They also studied the fate of embryonal fragment grafts and their influence upon the survival rate and the incidence of neoplasms in the irradiated animals. —*Hans Lewin, M.D.*